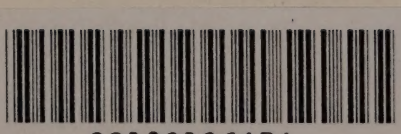
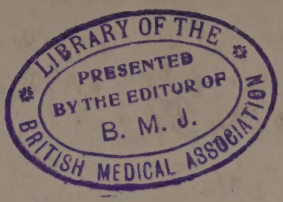
NEUROLOGICAL CLINICS

EDITED BY

JOSEPH COLLINS, M.D.

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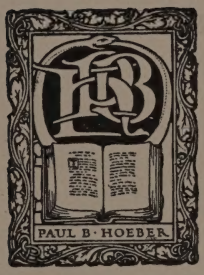
NEUROLOGICAL CLINICS

EXERCISES IN THE DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM

Given at the Neurological Institute, New York by the Staff of the First Division

EDITED BY

JOSEPH COLLINS, M.D.



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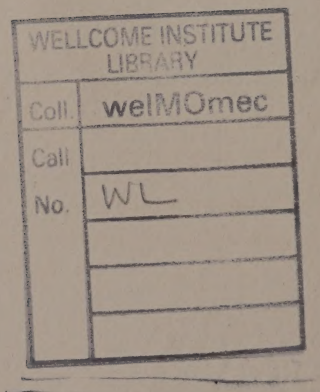

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WALTER CLARK HAUPT

IN MEMORIAM

The pleasure incident to the work which these pages represent was marred by the death of one of our most valued assistants, a young man of rare gifts and great promise. The satisfaction in doing it was steeped in despair, for we knew that soon he must die. It was he alone, though he shared our certain knowledge, who comported himself as though he would live the days allotted to man by the psalmist. He had been our intimate worker for two years. We had come to know his actual and potential possessions and we looked forward with confidence to their display, for the benefit of the sick and the poor, who made a singular appeal to him, and to whom he gave unsparingly his sympathy, his kindliness, and his talents. He had concealed from us only his surpassing courage, his marvelous equanimity. He took life joyously, a light in his eye and a smile on his face, and he went to death like an Olympian youth bounding over a hurdle.

Friendship with him was a privilege, collaboration an inspiration, coöperation an incentive.

Insatiabiliter deflebimus æternumque
Null dies nobis mærorum pectore demet.

LUCRETIUS

PREFACE

I venture to hope that the general practitioner as well as the neurologist will find the clinical lessons of this volume profitable reading, and that study of the examples of disease here depicted will be useful to him in the diagnosis and interpretation of cases which he encounters.

The cases are selected from the large number that are presented by the staff of the First Division of the Neurological Institute at their bi-weekly conferences. Care has been taken to select examples of disease that are most likely to be encountered by the practitioner and which he finds difficult to interpret and diagnosticate satisfactorily.

The plan followed is to state succinctly the chief features of the patient's ailment and to outline the picture as it developed, hoping that this alone will suggest the diagnosis. Then the objective symptoms are enumerated and some explanation made of the way in which they indicate the correct diagnosis. In some instances a brief discussion of the differential diagnosis is appended; in others reference is made to the prognosis and treatment. The chief object, however, of these lessons is to discuss the diagnosis. The intelligent, successful treatment of any disease presupposes a correct or an approximately correct diagnosis; hence, the stress that is laid upon it. It was once a popular fiction that no successful treatment of nervous diseases existed, but that was at a time when no nervous diseases save epilepsy and hysteria were recognized.

The present volume is the first of a series which the personnel of the First Division of the Neurological Institute hope to publish annually, should they find that it is of use to their colleagues.

May, 1918.

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NEUROLOGICAL CLINICS

BRAIN TUMOR, FOCAL VASCULAR DISEASE OF THE CEREBRUM, DISSEMINATED SCLEROSIS

JOSEPH COLLINS, M.D.

THERE are many organic nervous diseases that can be correctly diagnosticated, even in the early stages. In the main, they constitute the diseases of the spinal cord. Diagnosis of disease of the brain is largely a matter of approximation, that is, of guesswork. The person who has had experience in guessing is more likely to be right than a novice, but oftentimes the expert misinterprets all the signs. To be sure there are certain areas of the brain that have their definite functions—the precentral convolution, the angular gyrus, the middle of the left first temporal convolution, and so forth; but when such areas become diseased by chronic inflammatory processes, by tumor formation or by slowly developing vascular lesion, the symptoms that result may be the same in each of them and the clinical manifestations of irritation or destruction of such areas caused by one of these pathological processes are not to be distinguished from those caused by another quite dissimilar in nature. It, therefore, becomes necessary to distinguish them by other means, and here looms up a second difficulty. The general symptoms of cerebral disease have a close similarity, even those

caused by most dissimilar pathological processes. An approximation to the correct diagnosis can be made only by careful inquiry into the manner of development of the symptoms, their durability,—their transientness or permanency—their association with evidences of generalized disease (parasitic invasion, syphilis and malaria; arteriosclerosis; chronic nephritis; acidosis; sepsis; and so forth) and by awaiting the occurrence of symptoms that bespeak increased intraventricular pressure, especially swelling and edema of the papillæ of the optic nerves, or such well-defined indications of invasion as hemianopsia, aphasia, astereognosis, Jacksonian epilepsy or paralysis.

The importance of early diagnosis of diseases of the brain is emphasized by every one who encounters these cases frequently; yet, the melancholy fact remains that many of the diseases of the brain, especially new growths and vascular lesions not due to syphilis, are as unamenable to treatment applied early in the course of the disease as they are later. There are other reasons than therapeutic, however, for urging early diagnosis: it is gratifying to the diagnostician, satisfying to the family, and it may be the means of sparing the patient humiliating and painful experiences. Moreover, it often permits us to apply measures that shape the course of the disease to the comfort and temporal welfare of the patient.

Some of the difficulties of interpretation which many cases present are illustrated by the following narrative:

An Irish-American office worker, 46 years old, remarked in May, 1916, that he could not readily and effectually push aside objects—e.g., a cuspidor, wastepaper basket or chair—with the right foot. Within a fortnight the right leg felt heavy, as if he had to push it to walk, and he could not rely upon it either in standing or walk-

ing. Shortly after this he began occasionally to be incontinent of urine and feces. Save a few times, this amounted only to slight dribbling of urine and imperative desire to urinate. By July first, the right shoulder felt stiff and the patient noticed retardation in all movements —walking, writing, talking. He sometimes had difficulty in expressing what he intended to say and would only succeed after a patient effort. He experienced difficulty in writing—the grasp of the pen would often relax, the letters lost their symmetry and the script grew steadily smaller. Early in August he began to have involuntary movements in the right leg at night. They beggar description. He was not quite sure that he did not make the drawing-up jumping movement voluntarily as a response to the queer, indescribable feeling in the leg. Whether voluntary or involuntary the movement was quite uncontrollable, and night after night he would get up many times seeking surcease from it. About this time he noted a certain emotional instability. He would often burst into tears on encountering a trivial appeal to the sensibilities —a phrase in a letter, an expression of solicitude, a scene at the movies. He also realized that his mind worked much slower than was its wont. By the end of August throbbing pain in the left side of the head was added to the symptom group, also insomnia. The sense of immobility in the right leg had insidiously increased, and, though he walked quite well, he had to forego work. These symptoms did not materially change during the succeeding three months, save that he had less pain in the head than previously. The throbbing was replaced by a feeling of sensitiveness in the back of the head and neck on the left side.

Physical Examination.—His symptoms when first seen, October, 1916, were:

1. Impaired mobility, dexterity and strength on the right side.

2. Occasional incontinence of urine and feces.

3. Slowness of voluntary movements—writing, walking, prehension, talking (reduction of amplitude of these).

4. Tendency to emotional display independent of coincident feeling; slowness of mental process.

5. Jerking movements in the right leg, especially at night, associated with or preceded by a disagreeable sensation amounting to pain.

6. Throbbing pain in the left side of the head, more or less continuous, but subject to exacerbation.

7. Objective evidences of right side spasticity, exaggerated tendon-jerks, Babinski big toe phenomenon, Hoffman digital reflex, perseveration.

The objective symptoms that he presented are the customary accompaniments of spasticity dependent upon disease of the pyramidal tract, viz.: lively tendon-jerks, the Babinski big toe and Hoffman finger phenomena, diminished strength and impaired dexterity, while the perseveration is an indication of disorder of the extrapyramidal motor pathway.

His entire appearance was one of immobility. This was due in part to the fact that the right side of the face was not innervated so vigorously as the left, and the entire right side of the body was somewhat stiff. There was slight ataxia of the right hand, which was distinctly manifest when he attempted to write. The pupils were slightly uneven, the right being 3.5 mm. in diameter, the left 4.5 mm. The discs and fields were normal. There was slight, inconstant lateral nystagmus on looking to the right. The left occipital region and neck were sensitive to percussion and to deep pressure. The pulse aver-

aged from 65 to 75; the blood pressure from 110 to 120 systolic, 65 to 70 diastolic; the temperature was normal. Physical examination revealed no other abnormalities.

Such symptoms as this patient had may occur with three distinct diseases of the brain: tumor, focal vascular disease, disseminated sclerosis. Can we, with the data available, decide which of these diseases he has? The cardinal symptoms of brain tumor, namely, headache, vomiting, dizziness, unsteady gait, mental hebetude and the elevation of the optic papillæ do not exist. He complains of headache, but the pain seems to be an expression of the same condition as the tenderness in back of the head and neck, and it may well be that a local inflammatory condition in the connective tissue of the muscles of the aponeurosis might account for the pain. If it is unjustifiable to make the diagnosis of brain tumor in a case in which the cardinal symptoms do not exist, such diagnosis could not be made here. On the other hand, the symptoms indicate that he has a slowly developing lesion in the left hemisphere involving the pyramidal and extrapyramidal motor pathway far beneath the cortex. The diminished strength and impaired dexterity of the right members, the jerking movements in the right leg preceded by or associated with the disagreeable sensation in the leg amounting to pain, the phenomenon called perseveration, the manikin-like immobilization point with much certainty to the localization of such a lesion. The tendency to crying spells not dependent upon depression and the occasional incontinence of urine and feces are quite reconcilable with such a lesion.

The lesion, however, need not be a new growth; it may be a patch of sclerotic disease, an islet of disseminated sclerosis; it may possibly be an endarteritis with subsequent thrombosis and softening. Against the first symp-

tom are the patient's age, the progressive course of the disease and the absence of any other manifestations of disseminated sclerosis, particularly disorder of the abdominal reflexes. Disseminated sclerosis may show itself first in an individual as old as this patient. The manifestation of the disease may be confined to one-half of the body; an involuntary emotional display is not infrequently a conspicuous symptom. Nevertheless, there is something about the way in which the symptoms unfolded themselves in this instance, and about their grouping, which makes the diagnosis of disseminated sclerosis far less probable than that of infiltrating glioma or vascular lesion. Experience has taught us that focal arteriosclerosis of the brain may exist without the manifestations of vascular disease in other parts of the body, but in such instances there is to be elicited a history of infection—such as grip, pneumonia, or syphilis—of injury to the head or of repeated or prolonged debilitating experience. None of them can be elicited in this case. Evaluating these facts and taking them in conjunction with the absence of indications of arterial disease in other parts of the body, I am inclined to exclude focal vascular disease in this instance.

The patient has the appearance of an individual stricken with paralysis agitans. Other facts in favor of paralysis agitans are that he is Irish, of the age when the disease most often develops, displays slowness of all voluntary movements and has become rigid and immobile generally. His handwriting, moreover, is very suggestive of paralysis agitans—the letters are small and the tracings display distinct tremor. Were it not for the lively tendon-jerks, particularly the Babinski big toe phenomenon on the right side, and loss of control of the sphincters, which bespeak an organic disease of the central mo-

tor pathway, the diagnosis of paralysis agitans could not be excluded, but in the face of their existence it must be eliminated.

In the absence of any history of chronic purulent disease in any part of the body, such as the middle ear, the sinuses, the teeth, and of disorder of the leucocytes, the temperature or pulse rate, one is justified in assuming that the lesion is not an abscess.

Taking all these facts into consideration, the most likely diagnosis is of a slowly growing tumor in the cortex of the left hemisphere beneath the central fissure at its superior terminus. The nature of the growth can only be surmised. It is probably not syphilitic, since no indications of that infection show in the blood and cerebrospinal fluid, nor tuberculous, as tuberculous growths practically never occur in late adult life. There are no conspicuous indications of meningeal involvement; therefore, it is not likely that it is endothelioma. If the lesion is a tumor, the growth is probably an infiltrating glioma.

Subsequent History.—This patient was frequently seen and examined until August 10, 1917. The symptoms gradually increased. His chief complaints at that date were stiffness of the neck radiating down to the right shoulder; progressive inability to use the right arm, while the impairment of the right lower extremity had not increased proportionately; vertigo; occasional incontinence of feces and urine; exhaustion and prostration on comparatively slight effort; a feeling of motor unrest in the right lower extremity at night after retiring; and attacks of uncontrollable yawning and gaping. The objective symptoms had all become intensified—the ironed out, flattened appearance of the right side of the face, the passive rigidity of the right side of the body and the evidences of right-sided pyramidal tract involve-

ment had all increased. He could scarcely close the right hand and was quite unable to grasp the knife in cutting food, and so forth. The one remarkable objective sign which he presented was an increase of the abdominal reflex on the right side, that is, the same side that showed the pyramidal tract manifestations. This was a paradoxical finding and not readily susceptible of explanation.

The character of the vertigo was such that it was attributed to poor vasomotor equilibrium in so much as it usually occurred on sudden change of position, such as attempting to get up quickly from a recumbent posture. There was no demonstrable disorder of the heart; the systolic blood pressure was 125, diastolic, 80. Despite this, the blood vessels appeared to reveal on palpation a slight degree of fibrosis. The yawning and gaping which latterly developed are symptoms not infrequently associated with chronic arterial disease.

He has many involuntary crying spells, and the slightest emotional reference or appeal will set him off. He has his good days and his bad days; but the latter are increasing. At times he drops off to sleep very rapidly during the day, but he can be quickly and completely aroused. In addition to this, he has attacks of what appear to be mild dyspnea without effort, which last for a minute or two and then pass away. The motor unrest of the right lower extremity which has been referred to is described now by his wife as the movements which a person makes when unable to get a limb in a comfortable position. He draws it up, stretches it out, draws it up again, and so on. In the morning on arising there is often pronounced tremor of the right upper extremity and apparent general immobility. This is frequently accompanied by an uncontrollable yawning.

The progress of the case seems more consonant with a slowly progressing vascular lesion than it does with a slowly infiltrating glioma, but, as was said in the beginning, it is impossible satisfactorily to make a differential diagnosis. One would expect that, with the latter disease, there would be some very distinct evidence of increased intracranial pressure, but so far, now upward of eighteen months after the advent of well defined symptoms, no such indication has appeared.

This case is particularly instructive if taken in conjunction with the following case.

TUMOR OF THE MIDBRAIN WITH AUTOPSY REPORT

HENRY R. MÜLLER, M.D.

The fact that certain brain tumors at times grow to considerable size before they cause symptoms generally regarded as pathognomonic of brain tumors is perhaps worth emphasizing again. Particularly the gliomata, which often undergo early cystic degeneration, do not produce marked intracranial pressure until after they have existed a considerable time. The case reported in this paper is remarkable in this respect. It was only two months before the patient's death, or at most three months after the onset of any symptoms, that the first cardinal symptom of brain tumor appeared. Throughout the entire course of the disease the patient was free from headache, nor did the eye examinations at any time reveal papilledema. The process was characterized by a slowly progressing paralysis which, as the autopsy showed, went hand in hand with the progressive growth of the tumor.

The clinical features of the course of the disease are as follows: The patient, J. A. P., a printer, aged forty-six, consulted Dr. E. G. Zabriskie March 27, 1917, complaining of slight weakness of the left side of the body. This weakness, he claimed, came on a few days after he had struck his knee in a fall received while going upstairs in the latter part of February, 1917. Although the patient believed that there was some causal relation between this

injury and his present condition, it seemed probable that, even at that time, he had some slight weakness of the legs which he had not noticed and which was responsible for the fall. Moreover, his wife and family physician both maintained that for at least two weeks previous to this time, he had not been using the left leg with the same readiness and dexterity that he did the right. Soon after this he noticed a weakness in the left arm. In addition, his vision had been failing since the first of January. He had not vomited, nor had he had any nausea, headache or fainting spells.

Physical Examination.—On March 27th, the findings were those characteristic of a left spastic hemiplegia of face, arm and leg. His station was good, and there was no Romberg sign. The knee-jerks and arm-jerks were more active on the left than on the right. The left epigastric and abdominal reflexes were absent; those on the right were present. The Babinski phenomenon, ankle clonus and the Hoffman sign were all present on the left.

The left palpebral fissure was larger than the right. Mobility of the eyeballs was good. The left pupil was somewhat larger than the right, but both reacted promptly to light and in accommodation. There was no nystagmus.

The chest and abdomen were negative. The pulse rate was 76 and the blood pressure 130.

Laboratory Findings.—The Wassermann of the blood was negative. In the spinal fluid only a reduction with Fehling's solution was obtained. The Wassermann, the colloidal gold test, the cell count and the globulin test were negative.

Subsequent History.—About May 8th, he first noticed diplopia. Soon after this he vomited for the first time.

He had, however, no vertigo, but had had a queer sinking sensation associated with vomiting.

On June 11th he noticed a thickness of his speech, as though he could not articulate. It was only with some effort that he could make his speech distinct. Food would stick to the inside of the mouth and would have to be dislodged with the finger, but he did not notice that it was more marked on one side than on the other.

On June 18th the physical examination found his condition intensified since March 27th, but not otherwise materially changed. He was in full possession of his faculties, recollected the way in which his illness developed, and chronicled the approximate date of the appearance of each symptom. He had no ringing in the ears, no noises in the head, no disorder of hearing of which he was conscious, and no sensation of disequilibrium.

Objective Manifestations.—As he lay in bed the head was drawn over to the left and fixed. The left upper extremity was held semiflexed and fixed at all joints; the left lower extremity was slightly semiflexed and not as fixed as the left upper. The typical accompaniments of a left-sided organic spastic paralysis were present. There was a profound disturbance of deep sensibility throughout the entire left side, more marked in the lower extremity than in the upper. There was no distinct loss of tactile sensibility. The patient complained bitterly when an attempt was made to straighten the fingers, or any part of the upper extremity.

When not directing the gaze, the eyeballs both deviated to the left, and tended to remain in that position. This was not dependent upon a paralysis, because the patient could look to the right. There was no fixation paralysis and no inability to look upward. However, he neither focused nor looked upward with the celerity and

completeness of a normal individual. He had a distinct anesthesia of the right cornea, but not of the left. The left pupil was larger than the right. Both pupils responded promptly to light and through a fair arc. On looking to the right there were coarse nystagmoid movements which, if they persisted for any length of time, made the patient feel dizzy and nauseated. If the head was turned forcibly to the right, he vomited. There was a definite weakness of the right masseter and temporal muscles.

The pulse was slow, 68. The fundi were normal and there was no papilledema.

Up to July 9th, the time of his death, the patient became gradually weaker and more apathetic. From July 5th he was in a stuporous condition, but could be slightly aroused. His pulse became rapid and weak, and his breathing showed the Cheyne-Stokes phenomenon.

Autopsy.—Autopsy was performed three and a half hours after death. Only the brain was removed. The calvarium was normal. The vessels of the meninges and hemispheres appeared normal, as did also the hemispheres and the fissures themselves. A cystic mass was felt in the right middle cerebellar peduncle and pons. The right trigeminal nerve was pushed backward and outward, and was reddened and somewhat flattened. The right fourth, sixth, seventh and eighth nerves appeared normal.

After thorough fixation in formalin, the entire brain was cut serially into coronal sections. The midline of the brain bulged over to the left. The ventricles were collapsed. In the right cerebral hemisphere there was a cystic structure, with ill-defined limits, containing thick, yellowish, jelly-like material. The anterior pole of this cyst was situated approximately as far forward as the

anterior tip of the temporal lobe, and ran longitudinally backward toward the occipital lobe, destroying in its course the internal capsule and the lenticular nucleus. The cavity was about one and a half inches in diameter and three and a half inches long. The caudate nucleus and the optic thalamus were not destroyed, but formed the mesial boundary of the cyst. This tumor mass extended down the crus into the pons, being here more solid and mottled with numerous hemorrhagic areas. Its limits were difficult to make out, but the tumor tissue appeared to be more abundant on the right side than on the left. As it proceeded backward, along the floor of the fourth ventricle, it was represented by a narrow reddish zone along the midline. It finally became indistinguishable at a point about opposite the origin of the sixth nerve.

Epicrisis.—In view of the autopsy findings and the chronological events in the patient's history, it is easy to conceive that the tumor had its origin around the posterior portion of the internal capsule, and from there progressed forward, destroying the anterior portion of the capsule, and backward through the crus into the pons, and along the floor of the fourth ventricle. Weakness and paralysis of the leg came first, followed by weakness in the left arm. The tumor mass in the internal capsule correspondingly appeared to be the oldest portion, for it had here undergone most advanced cystic degeneration. In its extension into the pons the tumor destroyed or paralyzed the nuclei of the third, fourth and fifth cranial nerves. The neoplasm here was more firm and seemed to be the most recent.

Although on the one hand the total quantity of tumor tissue produced was considerable, on the other hand there was a rapid extensive destruction of tissue with

cyst formation, and absorption of material, so that the intracranial pressure at no time was very appreciably increased. Hence, there were prominently present the symptoms dependent upon the destruction of brain tissue, i.e., localizing symptoms, and only late in the course of the process were there present the few symptoms due to intracranial pressure, as noted above.

A CASE OF SUPPOSED BRAIN TUMOR HAVING AMONG OTHER SYMPTOMS A PARALYSIS OF COMITANT SINISTROVERSION OF THE EYES

WARD A. HOLDEN, M.D.

A cortical association center for turning the two eyes to the left lies in the right central frontal convolution. From this *cortical center* tracts pass to the *pontine center* for turning the eye to the left, namely, the left abducens nucleus. The left abducens nucleus is connected through fibers running in the posterior longitudinal fasciculus with the nucleus for the branch of the third nerve supplying the right internal rectus. Hence destruction of the left abducens nucleus abolishes movement of both eyes to the left beyond the median line.

In cases of apoplexy a frequent symptom is conjugate deviation of the eyes to one side with turning of the head to the same side. In such apoplectic cases probably the cortical center of ocular version or the supranuclear association tract is involved, but the resulting deviation lasts only a few hours at most. A permanent conjugate deviation of the eyes, however, almost invariably means a disturbance of the pontine center, the abducens nucleus.

With destruction of the abducens nucleus there is not infrequently associated an involvement of the homolateral facial nerve which curves round the abducens nucleus. There may also be compression of the pyramidal tract fibers which lie ventrally to the abducens nucleus.

In the present case the homolateral facial nerve is spared, but the pyramidal tract is involved. Furthermore, there is involvement of the contralateral facial nerve, indicating either a very diffuse lesion or multiple lesions. Details of the case follow.

M. L., aged 30, came to the Neurological Institute June 28, 1917, complaining that four weeks before, while at work, he had been seized with severe frontal and occipital headache, numbness of the right arm and leg and weakness of the right leg. Vision was confused. Three days later he had diplopia. After a week tinnitus was noticed in the left ear and slight difficulty in swallowing—fluids passing into the nose. He grasped objects uncertainly and tottered while walking. His wife had noticed a slowness of speech.

Physical Examination.—1. Eye—Corneal sensibility normal. Pupils—R. 4 mm., L. 3.5 mm.; irregular, slightly sluggish to light, good convergence reactions. No nystagmus, intermittent diplopia. Inability to turn either eye to the left beyond the median line, except in efforts at convergence when the right eye could be turned in slightly. V=20/20 each. Fields and fundi normal.

2. Slight weakness of right side of face (supranuclear).

3. Deflection of tongue to right.

4. Slight weakness of right arm and leg.

5. Unsteady station and gait with a tendency to fall to the right, slight incoördination of right hand (adiadochokinesis).

6. Lively tendon reflexes, the right greater than the left. No clonus, Babinski or Oppenheim signs.

7. All forms of sensations normal, though at first apparently diminished on right half of body.

Laboratory Findings.—Blood Wassermann and spinal fluid negative.

Subsequent History.—July 2, 1917. General condition worse. Could not walk without assistance. Severe headache. Nausea but no vomiting; no papilledema; labyrinths normal.

July 9, 1917. Intermittent vomiting for three days. Severe right headache and pain in right arm. Contracture of the left internal rectus now prevented the left eye from being turned outward to the median line. No papilledema.

July 11, 1917. Considerable mental deterioration. Progressive bulbar speech indicated an extension of the growth to cause involvement of the ninth cranial nerve.

We felt justified in assuming, therefore, that we were dealing with a rapidly growing tumor situated in the pons beneath the ventricle, which had already destroyed the abducens nucleus and which was causing symptoms of increased intracranial pressure possibly by intraventricular distention. The cerebellar symptoms might readily be explained by assuming an irritation of the superior cerebellar peduncles, and the symptoms of implication of the ninth nerve by extension of the lesion downward. In the absence of the smallest indication that the lesion was of a syphilitic nature, no treatment was given.

A CASE OF BRAIN TUMOR WITHOUT CHOKED DISC UNTIL ONE WEEK BEFORE DEATH

SANTE NACCARATI, M.D.

Choked disc, as a symptom of intracranial growth, is found at some time in the course of 90 per cent of the cases of brain tumor. Indeed, so constant is choked disc in brain tumor that its presence always suggests the diagnosis, although it is found also in various forms of meningitis, in brain abscess, in lues cerebri, in hydrocephalus and occasionally in chlorosis, nephritis and lead poisoning. It should be borne in mind that though the ophthalmoscope is a most valuable help in diagnosis of brain tumor, the ophthalmoscope should not be relied upon to make the diagnosis. The neurologist, not the ophthalmologist, should make the diagnosis; for the neurologist who would expect a choked disc in all brain tumors, and who would hesitate to make the diagnosis of brain tumor, notwithstanding the other general and focal symptoms, only because he did not find a choked disc, would not infrequently be mistaken.

The choked disc may be absent at the beginning of cerebral tumors of any kind and may continue absent during the course of small growths, cystic growths, diffuse extracerebral growths, in tumors of the pons, or of the medulla oblongata, and also of the motor area such as the case herewith presented.

The patient, an Italian, 55 years old, by occupation longshoreman, complained of gradual loss of power in

the left upper extremity and weakness in the left lower extremity. Eight weeks ago he was working at the dock during a very cold day and, while attempting to take a rope, noticed that he could not grasp it with the left hand. This state lasted only a few minutes.

One week later at 9 A. M. he was suddenly seized by an attack of jerking in the left upper extremity, which lasted about three hours, after which the left lower extremity and the left side of the face took part in the jerking and he fell to the ground unconscious. He was unconscious for about twenty minutes, after which he slept. The next morning he was able to get up and go into the street without any external appearance of what had happened the previous day. He tried to connect this happening with the influence of the extreme cold and the high temperature of a stove, to which he exposed himself alternately the preceding day.

Six days later he had another attack of the same kind lasting half an hour without loss of consciousness; this was followed by another nine days later. A few days later he noticed paresthesia in the left hand and that he could not button and unbutton his clothes. Four or five days after this, while he was eating, he suddenly lost the power of his left hand (the patient was left-handed) so that he could not keep the fork in it. This condition lasted only two or three minutes.

Until this time he had been able to walk and to perform all other movements with the hands and feet. Afterward he noticed increasing weakness of the left upper and lower extremities which rapidly progressed until, at the time he came to the clinic, it affected his gait and interfered with the movements of the left upper extremity.

During the last three or four weeks he had also expe-

rienced attacks of vertigo and headache in the right frontoparietal region. The headache gradually increased in intensity and was followed by a state of mental dullness amounting to stupor.

Physical Examination.—The physical examination made before and after the appearance of the left-sided paresis showed the following condition: The superficial reflexes of the left side were absent, the tendon-jerks on the left side were greater than on the right. There had been no big toe phenomenon during the first six weeks, but after that the Babinski sign was markedly present on the left side. There were left facial weakness and a slight left exophthalmus. There was no clonus, no Hoffman, no nystagmus. The pupils reacted to light and in accommodation but they were slightly irregular in outline. The left cornea was anesthetic and a left homonymous hemianopsia was found. The fundi were normal.

Besides, before the state of paralysis was reached, on the left side there were tactile agnosia, astereognosis, asymbolia, loss of sense of posture in the left foot, ataxia, and a suggestion of alexia and apraxia. The blood pressure at the beginning and later was between 140 and 148; the cardiorenal condition was fairly good.

Laboratory Findings. — Phenolsulphophthalein test gave 60-65 per cent of reduction. Two Wassermann tests of the serum were negative. These tests excluded in a satisfactory manner softening and other vascular lesions such as a toxic and a luetic thrombosis. There had been no vomiting, no bradycardia.

Diagnosis.—The diagnosis of brain tumor is by all means the most probable, notwithstanding the absence of choked disc. Making a minute analysis of the direct focal symptoms and of the neighborhood and distance symptoms, the site of the growth was located at the mid-

dle third of the right gyrus centralis, anterior and posterior, with invasion of the gyrus supramarginalis and of the lobulus parietalis inferior.

The rapidity with which the symptoms followed during the next two weeks may be explained by assuming that the tumor was a glioma, and that a hemorrhage had occurred either into the substance of the growth or into the surrounding area.

The short duration of the illness, eight weeks, does not exclude tumor, as it is known that many times a neoplasm may grow without any symptoms and, remaining latent for a considerable length of time, may later manifest itself suddenly and violently in the most tragic manner.

An operation was advised and accepted by the family. The patient's condition became rapidly worse. He had been stuporous the day before and during the day set for operation. He was brought to the operating room, but, since the surgeon feared that the patient would become aphasic for the rest of his life and was, therefore, willing to perform only a decompression on the left side, it was decided not to have the operation. The patient was sent home where he was considered in a hopeless condition for two days. At the end of the third day a slight improvement was observed. After one week he was able to move the left leg and touch the chest with the left hand; after two weeks he could raise the left hand to the nose.

Ten days later I found a well defined choked disc (6 diop.). It was proposed that he return to the hospital in order to attempt the operation, but the family refused, thinking that the patient would continue to improve, as they felt assured that no tumor existed.

One week later, however, he was seized with a convul-

sive attack of the whole left side, after which he remained completely paralyzed and gradually fell into a comatose condition. He died the following day. Consent for autopsy could not be obtained.

CEREBELLOPONTINE ANGLE TUMORS

GEORGE J. WRIGHT, M.D.

Tumors ordinarily found in the cerebellopontine angle are fibromata. They have their origin as a rule from the sheath of the eighth nerve. They are of slow growth, non-infiltrating, and the pressure symptoms which they cause display themselves as a fairly definite syndrome. Not all tumors in this locality arise from the sheath of the acoustic nerve. A few arise from the sheath of the fifth, seventh or ninth nerves. The tumor may take its origin not from a nerve at all but from the nervous tissue of the pons and cerebellum and the connective tissue of the meninges and blood vessels, as a glioma, a sarcoma or an endothelioma.

The diagnosis of tumor at the pontocerebellar angle may or may not be easy. It is unwarrantable to refuse to make the diagnosis because some one symptom considered to be pathognomonic is absent, such as, for instance, evidence of involvement of the acoustic. In the case reported herewith the symptoms for a long time were those of the fifth and seventh nerves before any other symptoms appeared.

A saleswoman, single, 47 years old, was seen May 29, 1917. Her chief complaint was twitching of the left side of the face and eyelid, accompanied by a drawing sensation. There was pain all over the head and in the neck, but not in the face. In walking she felt dizzy and tended to fall toward the left. The symptoms began when she

was 44 years of age with a sensation as if sand or an eyelash were in the left eye, and a little later as if a bug were gradually crawling down the left side of the face. Two or three weeks later, twitching began in the left eyelid and gradually involved the whole left side of the face. There was no pain or headache until April, 1917, when pain was felt in the back of the neck, in the head and in the temples. This was more a soreness than a headache and was usually worse toward evening. About ten days before the patient was seen, dizziness began, and since then she had felt as if she were in a swing and moving toward the left. This feeling disappeared when lying down. In walking she was so unsteady that she needed assistance. She had never seen double, but she thought her eyesight had become weaker. The back of the tongue felt numb and she had a feeling in the tip as if it were burned, and there was a constant sweetish sensation in the mouth. At times there had been roaring, singing and whistling in both ears but this was not constant—troublesome. She thought her hearing was good. There was no history of vomiting, convulsions, unconsciousness, weakness of the arms and legs, bladder or rectal disturbance. These subjective symptoms, therefore, arranged in the order of their development, were as follows:

1. Ophthalmic paresthesia.
2. Superior maxillary paresthesia.
3. Facial spasm.
4. Headache.
5. Vertigo without diplopia.
6. Lingual and pharyngeal paresthesia.
7. Occasional roaring in the head.

Physical Examination.—The chief objective symptoms found after repeated examinations were:

1. Ocular—Upward and lateral nystagmus.
2. Fifth nerve—
   (a) Sensory—definite diminution of touch, pain and temperature sense in all three branches of the fifth nerve on the left side.
   Loss of corneal and sneezing reflexes on the left.
   Loss of taste on left side of tongue.
   (b) Motor—Weakness with atrophy of the muscles of mastication on the left side. The jaw deviated distinctly to the left.
3. Seventh nerve—Paresis and irritation in all branches. The spasms were paroxysmal of varying intensity, tonic and clonic in character, and often drew the face violently to the left.
4. Eighth nerve vestibular branch—Vertigo in the erect position and subjective sensation of movements to the left.
5. Ninth nerve—Loss of taste on the posterior third of the tongue on the left with diminished or absent pharyngeal reflexes on the left.
6. Cerebellar—Unsteady station and markedly ataxic gait with a tendency to fall to the left. No adiadochokinesis.
7. Pyramidal tract—Slight increase in the right knee-jerk without clonus or Babinski.

The eye examination showed the left pupil slightly smaller than the right, nystagmus, no diplopia and normal fundi and visual fields. The ear examination showed both acoustic and static labyrinths normal to tests. Lumbar puncture was done and the fluid found to be negative.

Subsequent History.—A suboccipital operation was performed by Dr. Elsberg who found a small elongated

fibromatous tumor attached to the fifth nerve and extending downward in the cerebellopontine recess. The tumor was removed in great part. The patient made a good operative recovery.

The more one can convince himself, from consideration of the way in which the symptoms develop, that the eighth nerve is the first to be involved, with later affection of others, the more favorable will be the prognosis for complete removal. If the symptoms are not definitely of the acousticus type the less certain will be the chances of its successful removal. It must be remembered that angle symptoms may be remote pressure symptoms of a tumor in the vermis, in the homolateral, and also in the contralateral hemisphere. Spiller and, recently, Cadwalader have drawn attention to the fact that tumors may arise from the vicinity of the sensory root of the fifth nerve in the posterior fossa or from the Gasserian ganglion in the middle fossa. In two of the nine cases of angle tumor reported by Cadwalader the tumors were endotheliomata beginning in the region of the Gasserian ganglion and displaying symptoms different from the usual angle tumor. These tumors, arising from the dura at the base of the brain in the middle and posterior fossæ, frequently involve the bones of the skull, and do not infiltrate the brain substance but embrace the cranial nerves. As flat slow growing masses, they may extend forward or backward. In the latter case they involve the seventh and eighth nerves and produce symptoms resembling true acousticus tumors. But these tumors must be differentiated from acoustic tumors before they are submitted to the surgeon, because the operation for their removal is lateral, while for the acoustic tumors it is suboccipital. The most important differentiating symptoms are pressure or irritative manifestations in the domain of the sen-

sory fifth nerve, associated with third nerve paralysis, disturbance of smell, unilateral visual disturbances, and ataxia. These symptoms develop before deafness. Indeed deafness may be absent. When the tumor begins in the posterior fossa, it presses on the sensory root of the fifth nerve and grows backward. The differentiation then is most difficult. Tumors arising from the sheath of the acoustic are characterized by involvement of the fifth, sixth, seventh, eighth, ninth, and tenth nerves, the fifth and eighth nerve symptoms predominating in the beginning.

Angle tumors are usually slow in growing, and the first symptoms may be those of increased intracranial pressure, in which headache and papilledema are early and severe. This, however, is the exception, not the rule. The later symptoms are a combination of pressure and irritation on the cranial nerves, the cerebellum and the brain stem. The symptoms of palsy predominate over those of irritation. Impairment of hearing, and subjective sensations of buzzing or roaring are among the earliest and practically constant signs. Later there may be partial or complete paralysis of the muscles of one side of the face, neuralgic pains, paresthesias, absence of the corneal reflex and finally hypesthesia with weakness of the muscles of mastication. The glossopharyngeal and the vago-accessory nerves may be affected, and rarely the twelfth nerve. The motor nerves of the eye are seldom involved, although occasionally the sixth is affected. Pressure on the middle peduncle and the lateral lobe of the cerebellum is common and produces ataxia, nystagmus and at times adiadochokinesia. Pressure on the pons and medulla may be displayed by symptoms of pyramidal tract irritation. Positive localizing data, however, are furnished by the fifth, seventh and eighth nerves, and

final judgment is based chiefly on the consideration of the degree of involvement of these nerves. Evidence of definite impairment of any one of them is indicative of a localization of a tumor on that side. The localization is complete when there is involvement of either or both of the other two nerves on the same side. Excellent summaries of the symptomatology of cerebellopontine angle tumors have been made by Henschen, Stewart and Holmes, and Weisenberg, and more recently by Grey, who had available sixty-three cases of subtentorial tumors occurring in Cushing's clinic. In nineteen cerebellopontine angle tumors, Grey found no affection of the fifth nerve in four cases. The cutaneous disturbances were homolateral in ten cases, contralateral in one case and bilateral in two cases. The corneal reflex was absent on the homolateral side in six cases, on the contralateral side in one case and bilateral in three cases. Motor weakness was found in four cases homolaterally and contralaterally in one case. Cadwalader in a study of nine cases found subjective sensory disturbance mentioned in two cases and both subjective and objective in one. In three cases there was motor weakness. Henschen found incomplete involvement of the fifth in 70 out of 80 cases, but in only 16 was there actual anesthesia. In 20 cases there was involvement of the motor fifth. The sensory fifth nerve should be carefully examined in all cases, especially with respect to epicritic and protopathic sensibility and the condition of the corneal, nasal and palatal reflexes.

Grey found the seventh nerve affected in six out of 19 cases. There was homolateral involvement in eight cases and in five there was questionable involvement. In Cadwalader's report of nine cases, the facial was affected in six. Henschen in 100 cases found seventh nerve

impairment in 85, and of these 70 showed paresis homolaterally and 10 contralaterally. The almost constant type of seventh nerve trouble is some degree of palsy of the peripheral type. Irritation phenomena may be displayed by twitchings or by clonic and tonic spasms of a most pronounced type. Cushing has recently reported two cases in which the spasms were severe and persistent. In one a diagnosis of focal cortical epilepsy had been made. Further study and operation proved both to be due to a peripheral irritation of the facial nerve by tumor. While occasional fleeting twitchings have been observed and reported by many, Cushing states that in a series of 50 cases of tumor this particular irritative symptom in corresponding degree has not been observed. Henschen reports the occurrence of irritative symptoms on the same side in ten cases out of 100. Stewart and Holmes state that twitching of the homolateral side of the face occurs in cases of extracerebellar tumor, presumably owing to direct irritation of the nerve. Two cases similar to Cushing's have been reported by Weisenberg, Weisenberg and Mills, and one case by Spiller.

Involvement of the eighth nerve in cerebellopontine angle tumors is the most frequent of all. In Grey's series neither nerve was affected three times. In fifteen patients there was marked impairment on the homolateral side and in one patient slight impairment. Tinnitus was observed in twelve cases. In Cadwalader's cases the eighth nerve was involved in all but two and he concludes that nerve deafness is one of the most reliable localizing signs, and that unless it occurs at some time during the course of the disease, the diagnosis may be considered as somewhat uncertain. Henschen in 115 cases reports some eighth nerve affection in all but two, and in one of these there were auditory hallucinations and in the other early

tinnitus. That a large tumor may be present in the cerebellopontine angle without causing trouble in the eighth nerve is exemplified in a case reported by Weisenberg which had been diagnosed as tic doloureux and the ganglion removed without relief from pain. The real trouble was not revealed until the autopsy, which showed a tumor attached to the motor and sensory roots of the fifth nerve and growing back in such manner as to separate the seventh and eighth nerves. Deafness had not occurred, and only a year before death there appeared some twitchings in the lower part of the face with evidence of weakness in the upper part. Cushing has remarked that the fifth and seventh nerves are capable of an astonishing degree of stretching if the tumor is slow growing; nevertheless this case of Weisenberg's is remarkable.

In the diagnosis of tumor in the cerebellopontine recess the importance of a proper estimate of the disturbance of the fifth, seventh and eighth nerves in the presence of signs of increased intracranial pressure has been emphasized. Only rarely can we be sure of our diagnosis when the disturbance is found in one nerve. If there is anesthesia dolorosa in the distribution of the sensory branch of the fifth nerve, a diagnosis of tumor of the sensory root or the Gasserian ganglion can be made. If there is great impairment, or loss of hearing in one ear due to nerve involvement, a diagnosis of tumor is likely. The whole question has been well summarized by Grey and it may be well to quote some of his conclusions:

"In subtentorial tumors involvement of the fifth cranial nerve has no topographic importance in diagnosis unless the tumor lies in one or the other cerebellopontine angle. Such a localization is likely only when the homolateral eighth or seventh nerve is also affected."

"A paresis or a paralysis of one facial nerve in tumors

of the posterior cranial fossa is strong presumptive evidence of the side of the lesion. When the eighth or the fifth nerve of the same side is also affected the diagnosis of a homolateral growth may be made."

"In subtentorial new growths a slight unilateral impairment of hearing which has appeared for the first time in company with general pressure symptoms is indicative either of homolateral tumor or less frequently of a median growth. When hearing under similar circumstances is greatly impaired or lost in one ear it points to a homolateral extracerebellar localization of the tumor. Such a diagnosis is confirmed when the seventh or fifth nerve of the same side is also affected."

SUDDEN DEATH IN A CASE OF UNLOCALIZABLE BRAIN TUMOR

J. L. JOUGHIN, M.D.

The patient, a female, aged 54, had always been in good health until the onset of the present illness, May, 1916. Her past history was negative in so far as it related to her present condition.

The first symptom was slight unsteadiness of gait which at first incommoded her but slightly. This condition slowly progressed until she became wholly unable to walk. A few days subsequent to the development of this difficulty in locomotion the second symptom developed, an intermittent tinnitus of the left ear, a manifestation which still persists. On June 15th she was first seen by a physician and from that time until July 29th no very definite change in her condition occurred although locomotion became increasingly difficult. On this latter date she was seized with a violent attack of vomiting accompanied by severe pain in the epigastrium. This was the first of a number of distressing attacks similar in character except that they were unaccompanied by gastric pain. These crises, ordinarily matutinal, were apparently induced by the ingestion of food and were often preceded by nausea. Only on one or two occasions was the vomiting projectile in character. As the disease progressed the attacks gradually increased in frequency.

Late in July or early in August her acuteness of vision

began to diminish, but as late as the first week in September she was still able to read the smaller type of the newspapers. After that time vision rapidly decreased until she could read only heavy block letters one centimeter in height, with extreme difficulty.

During September she complained of severe, lancinating, paroxysmal headaches localized between the eyes. Rarely were these headaches at the vertex or occipital. These paroxysms often preceded and were apparently relieved by vomiting. They gradually became more severe and more frequent, making life unendurable save in the interval between the paroxysms.

She lost weight progressively, probably largely due to the fact that she voluntarily refrained from eating, owing to the headache and vomiting which eating appeared to induce. Her mental processes became slow and the resultant difficulty of comprehension made it difficult for her to coöperate satisfactorily in the physical examination. Except for this retardation and a little occasional nocturnal confusion her mental condition remained unimpaired.

Physical Examination.—Station and gait were both impossible, as she fell when unsupported, though in no particular direction. In walking with support she slid her feet along the ground, taking always a very small step. All the tendon-jerks were normal. There was neither clonus nor Babinski. The cutaneous abdominal reflexes were present, but sluggish. Coördination of the arms and legs was unaffected. Sensation in all its modalities was apparently normal all over the body, although the patient's responses were not always completely satisfactory. Speech was slow but otherwise intact. The sphincters functioned well though on one or two occasions after entering the hospital there was urinary incontinence.

The cranial nerves were all intact. There were no signs of cerebellar involvement.

The eyes were normal save for a high degree of refractive error (5 to 6 diopters of hypermetropia) and the changes noted in the fundus. These fundus changes were papilledematous in character, more marked on the left side than on the right. Although the degree of swelling was slight the diagnosis of papillitis could nevertheless be made because of the obliteration of the normal disc outlines and the distinct congestion and slight tortuosity of the retinal veins. In order to exclude the possibility of this appearance being due to the marked hypermetropia the fundus was again examined. This second investigation confirmed the previous opinion that the changes observed were evidently symptomatic of increased pressure within the cranial cavity. Vision on the left was $\frac{20}{200}$, on the right $\frac{20}{40}$.

Laboratory Findings.—The serological and other laboratory investigations revealed nothing which could aid us in the diagnosis, the only abnormal feature being a pleocytosis in the cerebrospinal fluid of 20 cells per cubic millimeter. The systolic blood pressure varied between 110 and 130 millimeters.

Clinical Features.—It may then be stated that the patient presented the following noteworthy clinical features.

First.—The earliest symptom to develop was disturbance of locomotion which progressed rapidly and to such a degree that the patient could neither stand nor take a step without falling to the floor. There was no paralysis of the lower extremities and no incoördination to explain this. The first thought that comes to one confronted with such a condition is, are we dealing here with an hysterical manifestation, or astasia-abasia? The improbability of

it is great, but one does not see how it could be disproved in the beginning.

Second.—The vomiting preceded by nausea, induced by ingestion of food, not projectile in character and sometimes accompanied by gastric pains is not the type usually met with in conditions of increased intracranial pressure.

Third.—The marked visual defect present is concomitant with a very slight elevation of the nerve-head and there is a possibility that this appearance might be explained by the high degree of refractive error present. As is well known in conditions of this sort choked disc of high degree is often met with, the vision remaining almost entirely unimpaired.

Fourth.—The course of the disease was unusual. The patient was found dead in bed five days after presentation at the Conference and five months after the first symptom of the disease developed. She had been seen by the nurse fifteen minutes previously, who reported that she was apparently in the same condition as she had been throughout her stay in the hospital.

The rapid clinical course, the slight pressure symptoms with late onset, and the sudden death strongly suggest a gliomatous infiltration, the lethal termination being induced by a hemorrhage within the growth. Owing to the regrettable absence of autopsy no more definite statement can be made.

DISEASE OF THE MIDBRAIN

TUMOR OF THE PEDUNCULAR PORTION

JOSEPH COLLINS, M.D.

Organic disease of the midbrain is always difficult to diagnosticate, sometimes it is impossible, and often there is very little satisfaction in attempting thoroughly to delimit its confines. The truth is that in the majority of instances in which this part of the brain is diseased the lesion does not confine itself to one of the components of the midbrain, it usually affects or involves several of them simultaneously. We have had lately under observation and study here a series of cases, the variation of which may serve to illustrate how such diagnosis can be made, or at least how justifiable.

The first patient, a youth fifteen and one-half years old, applied at the Neurological Institute for treatment on March 27, 1912, complaining of weakness of the left hand and tremulousness of the left side, the arm and leg. His mother related that he had not been well for six years, that is, for five years previous to the beginning of these symptoms she noted that he was unlike himself. He was restless, irritable, complaining, troublesome, but there were no more definite symptoms until his fourteenth year, when she noted that he had difficulty in holding objects in the left hand, it trembled so much. When the hand was at rest there was no indication of tremulousness. Soon after this the weakness of the left leg

was noticed and he said that he found that it would often catch the ground when he tried to move quickly.

The next symptom to develop was the dropping of the right upper lid; diminution of vision was noted at this time. The third symptom was dysarthria. He related that his articulation at times was indistinct, at times quite normal. In other words, the dysarthria was variable. The other symptoms, viz., left hemiparesis, tremor and ataxia and right ptosis and ophthalmoplegia were constant. There were no symptoms of increased intracranial pressure, nor did the ophthalmoscope show any signs of beginning optic neuritis. He was not seen again until September 15, 1913. All the symptoms had increased in intensity but numerically they were as before.

Physical Examination.—Examination showed a big-boned youth, five feet ten inches tall, of striking facial appearance, which was conditioned by a partial right ptosis, right strabismus, oscillation of the left eyeball, large bulbous nose, thick protuberant lips, asymmetrical face, the left side being larger, and a head covered with coarse thick hair.

The gait was unsteady, of hypertonic hemiplegic type, and his station was likewise unsteady. There was a slow, rhythmical tremor of the left hand, and of the entire upper extremity. It did not increase on voluntary movement of the hand. The hemiataxia was very evident when he attempted the finger and nose test; he rarely succeeded in getting to within three inches of the tip of the nose.

The tendon-jerks were increased on the left side of the body, more particularly the knee-jerk. There was a left Babinski but no clonus. The cutaneous reflexes were quite normal and equal on both sides.

Examination of the eyes showed corneal sensibility normal. Right pupil 4 mm., round, very sluggish reaction

to light and convergence. Left pupil 2½ mm., sluggish to light and in accommodation.

The right eyeball could not be moved upward, downward or inward, and there was almost complete ptosis. The mobility of the left eye was approximately normal.

There was slight nystagmus in lateral direction.

The optic discs were pinkish. The retinal veins were dilated and tortuous, suggesting beginning papilledema.

Vision with glasses was right 20/100, left 20/70.

There was a definite weakness of the left side of the face more marked in the lower than in the upper; this disparity in the two sides of the face was more noticeable on occasional display than it was on voluntary effort. The tongue protruded slightly to the left, but the palate was lifted on phonation equally. The palatal reflex was very much diminished.

Subsequent History.—The patient was not seen again until September, 1915. His symptoms had increased in severity. The external and internal ophthalmoplegia of the right eye was now practically complete. The left facial palsy was quite distinct; the left hypertonic hemiparesis was decidedly pronounced (though he was able to move about unaided). The eyes now showed marked papilledema, the left more distinctly than the right. The laboratory examinations of the blood, cerebrospinal fluid and excreta were at all times negative.

The patient was admitted to St. Luke's Hospital September 29, 1915, and died there November 14, 1915. Dr. S. W. Lambert, physician in charge of the service to which he was admitted, has kindly shown me his notes.

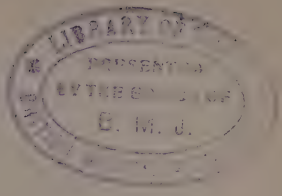
The only additional symptoms that the patient developed were headache, which became severe a short time before his death, and projectile vomiting. The phenom-

ena immediately preceding death were those of respiratory failure.

In brief, then, the cardinal features of this case in the order of their development in an acromegalic individual were:

1. Restlessness, irritability, change of personality.
2. Right ophthalmoplegia. Left hemiparesis, ataxia, tremor, facial paralysis.
3. Optic neuritis.
4. Headache and vomiting a few weeks before death.

In other words, the symptoms constitute the so-called Weber Syndrome, which is always due to a lesion of one cerebral peduncle. The clinical features of the disease, its course and outcome show the lesion to have been a tumor.



PROGRESSIVE HEMIPLEGIA

JOSEPH COLLINS, M.D.

We encounter many cases of intracranial disorder that require protracted observation before we can say with approximate correctness what the nature and location of the lesion is. Such a patient is herewith presented.

He is a single man, 36 years old, by occupation a broker, who was apparently in good health until the middle of September, 1916. He then noted that the right hand and arm had what he calls a "funny feeling," a feeling of numbness and impaired dexterity. This gradually increased to such a degree that within two or three weeks from the time he noticed it, the right hand became quite clumsy, and he would often drop things that he attempted to pick up or hold. He noticed particularly that he was unable to do an act to which he had long been habituated, namely, to take his watch from beneath the pillow in the morning while he was still lying in bed, in order to find out the time. Gradually his right leg became slightly unwieldy and he recognized particularly that he could not put his right foot down as firmly as formerly. This feeling of stiffness in the right lower extremity and the weakness of it were responsible for the feeling of uncertainty in walking that he had at times. The next symptom to develop was slowness of articulation. He noticed it first, he thought, about a month after the impairment of manual dexterity. Coincident with the onset of his symptoms he often had headache, frontal and occipital, but

it was neither severe nor constant. After these symptoms had been in existence about three months he began to have considerable vertigo, but he had difficulty in stating in a convincing way whether the symptom was really vertigo or a sensation of uncertainty in standing and walking, which might be dependent upon the partial hemiplegia. It is probable that it was more the latter than the former. All his symptoms were worse in the morning. For instance, when he first arrived at his office he would scarcely be able to grasp a pen or pencil. The pencil would wabble about in his hand and he would have to adjust it in the right hand with the left. As he continued to use the hand, however, it would become somewhat more agile.

He was first seen by Dr. Haupt, on January 15th, 1917. At that time he was complaining of the symptoms already enumerated and of violent headache, nausea and vomiting, the latter symptoms having been in existence only four or five days. They were of a character that we frequently see after lumbar puncture, and when it was learned that he had been submitted to this procedure a few days before, it was thought to be attributable to that.

Laboratory Findings.—The report on the cerebrospinal fluid and likewise the blood serum showed that there was no evidence of syphilitic infection.

He was admitted to the hospital on January 16th. The only thing of importance learned concerning his history was that when he was 19 years old a barber called his attention to a growth on the right side of the face. He went to Mt. Sinai Hospital, where it was removed under a local anesthetic. So far we have been unable to get a report on the nature of that growth.

Physical Examination.—Physical examination revealed practically no abnormalities, save a right-sided hemi-

plegia of mild degree and a blood pressure that was constantly considerably above the normal—the systolic being 165-170, the diastolic 120-135. The motor paralysis was more marked in the upper extremity than in the lower, and the face was distinctly involved. It had all the hallmarks of an organic hemiplegia, save one. There was no Babinski big-toe phenomenon. Another noteworthy feature was that the abdominals and epigastrics were lively, in fact, exaggerated on both sides. The digital reflex, often called the Hoffman sign, was elicitable in both hands, and on the right it was very marked, so that irritation of the nail of any finger produced a quick, sharp flexor response of all the fingers. There was no disorder of sensation either superficial or deep; he recognized the nature and uses of objects placed in the right hand. Aside from myopia, the ophthalmoscope revealed nothing abnormal.

He was kept in bed for a week and at the end of that time ceased to complain of headache and of nausea. He was then allowed to get up and go about the ward. The only complaint made was of occasional dizziness after eating. This would disappear on lying down.

He has now been in the hospital three weeks and there has been no change in his condition aside from the cessation of headache. He thinks his speech is somewhat improved, that is, he talks with less effort to bring out the words quickly. The right upper extremity is weaker than when he came. Objectively, there is no noteworthy change.

Diagnosis.—When the patient was first seen the suspicion was aroused that we were dealing with a brain tumor or with a syphilitic disease of the blood vessels, leading to partial obliteration of the left middle cerebral, and especially of the second or third branches of this blood vessel. Upon cessation of the symptoms which

might be construed to indicate increased intracranial pressure, namely, headache, nausea, vomiting and dizziness, the probability that we were dealing with a tumor became less; and when observation showed that there was no change in the optic discs, and no slowness of the pulse—the other common accompaniments of brain tumor—this diagnosis was no longer considered. The man denied syphilis, his cerebrospinal fluid was quite normal, the Wassermann reaction of the serum was negative and there were no objective manifestations of syphilitic disease; therefore, that diagnosis was not considered tenable. We were therefore led to consider thrombosis of the branches of the left middle cerebral as the most probable diagnosis. Thrombosis when not due to syphilis is due to atheroma or to changes in the blood; exceptionally, it is due to disease of the walls of the blood vessels caused by compression or invasion from without. The patient's blood was quite normal and the only indication of atheroma that he had was a moderately high blood pressure. There were no symptoms of generalized arteriosclerosis, but in establishing the probable diagnosis it was recognized that atheroma of the cerebral blood vessels may occur apart from sclerotic change of the blood vessels in other parts of the body.

Our chief object in discussing this case is to call attention to the fact that it is not unlikely that we are dealing with an example of a disease known as progressive hemiplegia and originally described by Dr. Charles K. Mills[1] under the title of "Unilateral Progressive Ascending Paralysis." Since that time Mills and Spiller have recorded two other cases with autopsies, in which there was found a primary degeneration of the pyramidal tract. In the cases that have been recorded by them the

[1] *Jour. Nerv. and Ment. Dis.*, April, 1900.

paralysis was of an ascending variety, that is, it began first in the leg. In one instance, at least, the paralysis did not confine itself to one-half the body. The patient eventually developed a triplegia. On autopsy, degeneration of both pyramidal tracts was found. It is, however, noted that in the patient herewith demonstrated, the symptoms showed themselves first and are still most conspicuous in the upper extremity. In every other respect he conforms very closely to the description given by Mills and Spiller.

The impression may be given that we are attempting an unnecessary refinement of diagnosis, but the development of the symptoms in this case is not that of cerebral thrombosis or softening due to atheroma; that may be the lesion in this case, but if it is, the lesion has produced symptoms quite unlike those which such lesions ordinarily produce. It is impossible to say positively at this time what the nature of the lesion really is, but this demonstration may serve to call attention to this valuable contribution by Dr. Mills.

HEMICHOREA SENILIS

GEORGE J. WRIGHT, M.D.

Acute chorea, so common in childhood, rarely occurs in the late years of adult life. This case is interesting because of the acute onset, the one-sided involvement, the short course (less than four weeks) and the prompt recovery under isolation and hydrotherapy.

The name "chorea senilis" is a misnomer, because choreic patients, even as early as the fourth decade of life, are clinically classed under this rubric. The first cases were reported by Graves in 1850 and later by Charcot. Oppenheim states that the common chorea minor can occur in old age and run its typical course, and that even hemichoreas have been observed. The disease is, however, rare. In 1893, in an analysis of one hundred and twenty-five cases seen at the Post-Graduate Clinic, Collins reported the maximum age to be twenty-eight years. Thayer in a study of eight hundred and eight cases treated at the Johns Hopkins Hospital and dispensary up to 1906 found only twenty patients over twenty years old and no specific mention was made of a case of chorea senilis. In 1900 Bischoff collected all the cases of acute senile chorea that had been reported, about seventy in all, including one described in detail by himself. Seventeen of his cases were in the fourth decade, fifteen in the fifth and twelve showed unilateral involvement. His conclusions were that senile chorea is an unusual disease, that men and women are equally affected, and that rheu-

matism and heart disease appear as unusual complications. The affection is double-sided in the majority of cases. In about 60 per cent of his cases the mind was not disturbed. Recoveries occurred in 20 per cent of the patients, in one year at the latest. The shortest course of recovery was two weeks in a woman aged seventy-five. Only a few autopsies were made and none of them indicated the cause of the disease.

The subject of this report, a married woman, fifty-one years old, applied for an admission to the hospital because of uncontrollable jerky movements affecting the left side of her body. The trouble began two weeks before, and grew rapidly worse so that the patient could not work at all and could walk only with difficulty. The arm was more involved than the leg and a few days before admission occasional twitchings appeared in the left side of the face. There was no pain, muscular weakness or disturbance of speech. The mental condition was normal. The patient had never had a previous attack of chorea or a rheumatic affection of any kind.

Physical Examination.—When first examined the striking symptom was the frequent, typical choreiform movements affecting the left arm and leg, and occasionally the face. The movements were sufficiently wide in range to disturb the arrangement of the bed clothes. Excitement and embarrassment, which the patient seemed to feel keenly, exaggerated the frequency of the movements. On examination the pupils were found equal and responsive to light, the tongue was protruded in the midline, the tendon reflexes equal and somewhat sluggish on both sides. Repeated examinations failed to show any evidence of involvement of the pyramidal tracts. Sensation in all forms was well preserved. The patient was a heavy, somewhat plethoric woman, whose teeth and

gums were bathed in the pus of profound pyorrhea. The pulse was irregular, but the heart sounds were fairly clear and without murmurs. The blood pressure ranged from 150 to 170. The urine showed albumin, a small amount of sugar and a few casts. The patient was put to bed, isolated, given an occasional dose of chloral, and put in a hot pack twice daily. Four days after admission to the hospital her improvement was striking. The movements had decreased about sixty per cent and the ataxia disappeared. Three days later the patient had fully recovered, and in a few more days she left the hospital, having been sick about twenty-three days in all.

Diagnosis.—This case in every way resembled the ordinary chorea minor occurring in childhood. Because of the presence of cardiovascular renal disease and the limitation of the movements to one side, one might assume that some temporary and fully recoverable vascular lesion had occurred in the cortex, the central ganglia or the cerebellum. Except for the choreic movements, however, there was no evidence of such a lesion and one must acknowledge as much ignorance of the real anatomical basis of the attack as in the ordinary case of Sydenham's chorea.

ASTEREOGNOSIS WITHOUT HEMIPLEGIA IN CEREBRAL ENDARTERITIS

EDWIN G. ZABRISKIE, M.D.

Astereognosis may be a symptom of disease of many different levels of the central sensory neurons. It is most frequently encountered as an ancillary manifestation of cerebral hemiplegia.

In the case herewith presented there were no indications of motor hemiplegia. The patient is a man, fifty-seven years old, who first came to the hospital in July, 1915, complaining of rheumatic pains throughout the right half of the head and body. Nauheim baths and high-frequency electricity readily relieved these symptoms. About a year later he again presented himself, complaining of the same symptoms, and treatment again proved efficacious. In 1913 he had had gangrene of the little toe of the right foot, which was cured in ten weeks. He was treated in the hospital by Nauheim baths and baking.

The patient again came under observation January 6, 1917, complaining of impairment of vision, loss of power and dexterity and of ability to feel with the right hand. There was no complaint of pain. These symptoms developed suddenly and were first noticed on January 1, 1917.

Physical Examination.—He was a short man, of small skeleton, good muscle tone and no atrophies. His teeth were poor and several old roots remained about which there was considerable pyorrhea. The heart, lungs and abdomen were negative, except for double inguinal her-

nia. There was no Rombergism and the gait was normal. The cranial nerves were normal. The right knee and biceps jerks were greater than the left, the ankle jerks present and equal. The abdominal reflex and the epigastric were livelier on the left than on the right side. Hoffman sign was present on the right. The pupils were equal, regular in outline and responded sluggishly to light, promptly in accommodation. Tactile sensibility was somewhat diminished over the palmar and dorsal surfaces of the right hand, curiously confined to the distribution of the median nerve. There was no disturbance of postural, pain or thermal sensibility. Astereognosis was complete in the right hand. The grip in the two hands was unequal, the left being stronger than the right. There was no tenderness of the nerve trunks or plexuses.

On admission to the hospital his blood pressure was 180, and during his stay from the 6th to the 26th of January it ranged from 130 to 180, the pulse from 64 to 96.

There was no difference in the radial pulses but the amplitude of the right radial seemed greater than the left, a moderate degree of fibrosis existing in both. The external iliacs were palpable, but neither the posterior tibials nor the dorsalis pedis was palpable in either foot.

On January 18th, articles placed in his right hand were identified as follows:

Key	Key
Safety pin	Round wire
Buttonhook	A pen, not a buttonhook
Watch	Watch
Penholder	Something long like a pen
Quarter	Money
Knife	Knife
Dice	A square thing
Nail	A ring, a round thing

This showed that astereognosis was far less pronounced.

The patient was discharged January 26th, as the complaints for which he entered had almost disappeared. Tactile sensibility and sense of position in the right hand were now about normal and he identified all objects correctly with the right hand. Treatment while in the hospital consisted of Nauheim baths, massage and high frequency electricity.

Subsequent History.—On February 8th he was reëxamined and showed still further improvement. Dexterity of the right hand had improved considerably. He complained occasionally of a sensation of heaviness in the head and of slight impairment of sight. In reading he frequently skipped a line and often began with the second or third word of a sentence.

Physical examination showed that the tendon jerks were alike on both sides and no Hoffman sign was demonstrable. There was slight hypesthesia of the tips of outer fingers of the right hand, more marked on the palmar surfaces.

In the right hand the following objects were recognized with little delay: Key, knife, pieces of money, pencil, chain, watch, match, matchbox.

Diagnosis.—The case impresses us as one of very slight lesion in the motor tracts of the left side of the brain with involvement of the adjacent sensory tracts. We are led to believe that the vascular system is at fault and that the lesion was a small area of ischemia due to arterial closure, subsequent probably to endarteritis with thrombus formation.

THE UNFOLDING OF A CASE OF APHASIA

C. BURNS CRAIG, M.D.

The occasion is not often afforded to study the manifestations or features of aphasia as they unfold; it is the customary experience to encounter an aphasic when the manifestations of his communicative disorder are fully developed. Hence the difficulty of studying such cases, and oftentimes, of interpreting them.

The patient whose case I present came to the clinic December 15, 1916. At that time he had some difficulty in externalizing mental concepts, both articulatively and graphically, but his speech was so fluent at times and his conduct so bizarre that he was looked upon by those who came in casual conduct with him as insane or simulating. Such suspicion was further heightened by the fact that his body was covered by small wounds, many of which had the appearance of being self-inflicted. His history is as follows:

The patient, a sign broker, single, 44 years old, attributed his nervous condition to business reverses caused by the rise in price of glass, which had interfered with his business prospects. The friend who accompanied him to the clinic stated that he had not been successful for a considerable time. He had been living alone in a furnished room and his associates had lost sight of him. He had always been a nervous, excitable man, but it was not until a few days before coming to the clinic that it had been remarked by those who saw him that there

had been a striking change in his speech and conduct. He gave his name and address promptly, but when asked his age, replied, "say, something, ah f f f 45-48-53 or something." "Don't you know your age?" "I tried my level best but cannot do it." "What is the matter with you?" "I don't know. It is just something. I cannot use this arm" (pointing to the right arm which apparently he uses with evidence of strength and dexterity). "Everything goes wrong. If I could only get rid of this—of this infernal business in my left arm here, no, wait, my right arm here." "What is wrong with your arm?" "Cannot use it. In the morning I cannot . . ." Then his voice grew louder, he smacked his lips, frowned, shivered and gesticulated despairingly, sighed and moaned, "My God, I cannot do it." He grasped his neck, sniffed louder, moved rapidly about the room and vociferated, "Dr. Wagner told me it would all come back." When asked to write his name, he took the pencil in the right hand and assumed the signatory attitude, but did not begin to write. After a brief time he threw the pencil down and exclaimed, "I cannot. It is all wrong." When asked to explain how he got the blisters (which are seen on the pulp of the fingers of the right hand) and wounds (which have the appearance of mild ulceration around the nails and on the back of the right hand as well as those all over the trunk and extremities which look like scratch marks made by the nails or some blunt object), his reply was, "I do not know. They must have come between Sunday and Monday."

It was learned that aside from a chancre which he had when 20 years old and for which he received very little treatment, that he had been in good health until the onset of the symptoms enumerated above. At times he used alcohol to excess. There was no evidence of paralysis in station, gait or in the use of either upper extremity. In

fact, the physical examination aside from slight elevation of systolic blood pressure, 165, was absolutely negative. He was admitted to the hospital and the next morning the supervising nurse reported that his conduct during the night caused a lot of disturbance in the ward. He was constantly getting in and out of bed, apparently searching for something that eluded him, talking to himself, groaning, making strange noises, picking at and removing the dressings of his wounds, etc.

Mental Examination.—The following is a stenographic interview with him on that date:

"When did you come here?" "Yesterday." "Who recommended you to this hospital?" "Dr. Wagner of Madison Avenue." "What did he say to you?" "He said there was something the matter, I could not lift this right arm, and I should come here and have something done." "What is the matter with your right arm? You seem to move it all right." "I do not know. All of a sudden, within a week to-day, or one or two days prior, all things came to me, this arm. The doctor said it would take me a week or two weeks to be treated for it." "Do you feel quite right mentally—do you think clearly?" "No, Doctor, I do not." "Can you describe how you feel, that is, can you tell me what you yourself realize? Your trouble —is it in thinking or in expressing yourself?" "No, I cannot describe it. I do not know how. I believe I am not just right. I am not sure, but I do not think I am just right." "What is the reason?" "I do not know." "Do you know where you are?" "Yes, sir; in the hospital." "Have you heard that you disturbed the other patients and that it would be necessary for me to send you away from here?" "No, sir; my God, what should I do?" "Are you without funds?" "No, sir; that is, I have funds; I want to get in shape." "Are your funds in a bank?"

"They are in. . . . My God, why can't I explain it?" "What bank?" "I cannot tell you which one. My secretary is attending to that." "What is your secretary's name?" "Schobow is the name." "A man or woman?" "My secretary is a man." "What nationality is he?" "I guess he is American, that is, he was born in America, but he was eight years abroad." "What is his address?" "I do not know; he will tell you." "What is the name of the man who came to the clinic with you yesterday?" "He is all right, a nice, fine man." "Can you tell me about these wounds on your right hand and body?" "I believe they may have come from Friday until Monday."

The excitability and irresponsibility of the patient were so great that he required narcotics at night and sedatives during the day. Within the next five days this state gradually subsided, but the disorder of speech steadily progressed. A typical conversation in the interim is as follows: "What is it you want to say?" "What I want to say is awful to say, but now there you are, if I could only say it. If I could only say that, that is all. If I could only say that I have mentioned this, nothing else, that is all. All I remember I wanted to tell you, I could not tell you about those people, because there you are." (Makes an effort with his entire body, grimaces, snaps his finger.) "I cannot say it." "Where are you?" "I beg your pardon, right here" (ceremoniously). "What institution is this?" "Sir? This place? Why right here, 90th Street, oh, I can't." "Do you know me?" "Certainly, I can't mention yourself, but you are very nil, oh God, I can't remember." "What is your name?" "What name?" (Thoughtful pause.) "Cannot do it, doctor." "Where do you live?" "90 and 19 and 70, can't make any difference, but that is the place, near Oak." "Where were you born?" "West. Sacramento. I was born in the South;

what difference one thing or another." "What city?" "Sir, I was visiting there a sister, then I came to the angle. Good God, I don't know what has come over me. I mean to say, but I can't do it." "Are you married?" "No, sir. Why? Is that final? Do you think so? I do not know what this is for me, I have had many things occur in my locket, and let her come. Since you said to me it does not look so much." "Repeat—Around the rugged rock." "Rucked down, around, upon ruckely bon, must, I cannot do it." "Repeat—Methodist." "Methodosal." "Say—electricity." "Electricity that's bro lost it again bru bigit losses big place clean frost." "Say—Massachusetts." "Mathew and one, oh! woun, cannot do it, in a few days." "Say—I can get agoing." "I would like to know what it is about." "Do you know what you want to say but can't say it?" (Excitedly) "Now you have got the whole ticket, Doctor, you know I have a lot of, you know it has got on my *wokedy.*" "Doesn't the word come to you?" "No." "Do you have an idea?" "Absolutely." At this period the patient showed no physical signs whatever and remained quiet for ten days.

On the 2nd of January, at the time of the morning visit, aphasia was nearly complete. The response to all questions was "Oh, oh, oh, oh, oh." There was a definite right-sided facial weakness, but no weakness of the tongue nor any vocal organ. The tendon-jerks were equal and normal. He was unable to write with either hand. He did not carry out certain simple commands, such as "touch your nose" or "touch your ear," but others were complied with, such as "open your mouth," "shut your eyes," "hold up your arm." On the next day, January 3rd, aphasia was complete, not even responses of "Oh, oh," could be made. He made violent grimaces and attempted to demonstrate his ideas

with his hands. There was a complete paralysis of the right side of the face and marked weakness of the right arm and leg. The patient confused simple commands—asked to whistle, he hummed correctly the first eight or ten bars of "The Toreador Song." On January 4th the right side of the face remained absolutely paralyzed; the right arm and leg were also now in a state of flaccid paralysis, with a Babinski sign. The patient showed by remonstrances that the sensation to pin prick was more pronounced over the entire left side than in the corresponding areas on the right. On January 5, 1917, motor and sensory aphasia were complete; complete right hemiplegia; involuntary urination. This state persisted for three days. On January 9th the right hemiplegia was less pronounced. He could raise up the right arm from the bed, but could not move the fingers. His replies to all questions were "Good God Almighty." He could not read script or print and the sensory paraphasia was evidenced by the following examples: "Put out your tongue." This he did. "Give me your hand." He again puts out his tongue. "Shut your eyes." He shut his eyes and put out his tongue. "Touch your nose." He protruded the tongue. "Hold up your arm." He put out the tongue. "Give me your handkerchief." He picked up the handkerchief and held it in the air, not toward the examiner.

From this time on, the paralytic state of his right arm and leg improved so that at the end of the month, although there was still evidence of spasticity, he could walk short distances and use his arm and hand with a degree of dexterity, although both arm and leg were weak. The weakness in the face was more pronounced. The motor aphasia continued fairly complete; one day he said "yes," but never again; on another occasion ut-

tered "all right," but the general responses continued to be "good" or "Good God Almighty." Effort at speech invariably resulted in a repetition of the same phrase in a louder voice. He still failed to perform correctly simple commands, spoken or written, evidencing thereby a degree of sensory aphasia. He could neither write nor read. The course of the systolic blood pressure is worthy of notice. It manifested a tendency to rise during the first five days, when it touched 180; during the next eight days there was a gradual decline to 110, then an abrupt rise to 165, at which time the complete aphasia developed. Following this there was a tendency to maintain a high pressure of 170 or 180 for six days, then slow declination again and the maintenance of an average of about 145 for over two weeks. From that time to the end of the observation, for about ten days, during which time he was occasionally out of bed, there was a tendency to slightly greater elevation. His pulse, which had averaged about 80, dropped to 50 preceding the complete aphasia and then moved up again, averaging 72 to 80.

Subsequent History.—After a lapse of seven months the patient was interviewed at the Montefiore Home. On entering the room, he manifested instant recognition of the examiner. His facial expression was animated and he tried in vain to express a verbal greeting, succeeding only in producing explosive vowel sounds.

The auditory sense of speech had entirely recovered. He performed rapidly simple commands and recognized the spoken names of objects correctly. He was unable to write or print but could read the words. For example, pen, pencil, chair, table, were set down and were pronounced with various other words. The patient was able to indicate each written word when it was spoken. Attempts at reply or spontaneous speech provoked a gen-

eral bodily effort, ending with a forced "Oh, oh," or "Good God." There had, therefore, been no change in motor speech defect, whereas sensory speech had improved greatly. Agraphia was still present.

AN UNUSUAL COMBINATION OF INVOLUNTARY MUSCULAR MOVEMENTS

EDWIN G. ZABRISKIE, M.D.

The subject of involuntary muscular movements, their relation to the basal ganglia and their connecting pathways, has in recent years received much attention, and, gradually, a clearer conception of the part these structures play in the maintenance of muscle tonus and in the pathogenesis of abnormal muscular states is being unraveled from the confused mass of evidence at hand. Our understanding of the part played by the corpus striatum and extrapyramidal tracts in the disturbances of primitive movements, associated movements and the causation of tremors, is due in a large measure to the brilliant works of Kinnier Wilson, C. Vogt, Oppenheim, Lewandowsky and others. Recently J. R. Hunt has attempted to establish a definite pathologic lesion in Parkinson's disease, and Jelgersma, Marburg, Touche and Maillard have also published causes of this disease with definite lesions.

Two regions have been found by these authors to be affected, namely: the lenticular nucleus and subthalamic fields of Forel, or the tegmentum and red nucleus. Unfortunately there is as yet insufficient corroboration to designate either of them at the definite location, disturbance of which will produce the Parkinson syndrome, although the preponderance of evidence seems to favor the lenticular nucleus. Maillard found lesions of the red nu-

cleus and tegmentum, but was unable to detect abnormalities in the basal ganglia. Touche and Marburg have reported similar findings. Jelgersma, on the other hand, using more improved methods, was able, in a case of senility and spasticity with a Parkinson tremor, to detect atrophy in the lenticular nucleus and external nucleus of the thalamus, but found the red nucleus and its vicinity completely normal. Hunt believes that definite cell layers in the lenticular nucleus are affected, but his findings still lack confirmation.

Kinnier Wilson has furnished, however, the most illuminating contribution to the subject in an exhaustive study of a group of cases of rhythmic, involuntary tremor, spasticity with contractures, emotional disturbances and explosive laughter dependent upon lenticular degeneration associated with cirrhosis of the liver. This work has opened wide the door to future investigation of the pathologic tremors and their combination with paralysis, muscular rigidity and the many types of emotional disturbances seen in organic disease of the central nervous system.

Interesting combinations of tremors occur occasionally, and although their record at the present time may not contribute towards the clearing up of the situation, I believe that the fact that they do occur is worthy of consideration. It is for this reason that the following case is analysed.

The patient, a Russian Jew, 53 years old, employed as a tailor, came to the clinic on June 5, 1916, complaining of impaired strength of the right hand, of "pins and needles" sensation in the hand and arm, and of diminished dexterity of the fingers which prevented him from working. He complained also of pain in the back of the head, under the right shoulder, and at times all over the

body. These symptoms, which had been in existence about three weeks, followed an attack of vertigo, which came on while eating dinner. He suddenly felt dizzy and fell to the floor; he did not lose consciousness but remained dizzy and uncomfortable all day. He went to work the four succeeding days; then the right upper extremity became weak. There was no real paralysis. A week after the first attack, and again while eating, he had a similar sensation of vertigo which, in varying degrees, has bothered him since. His family relate that his speech has become somewhat slower.

Physical Examination.—Examination revealed practically nothing aside from indications of chronic nephritis, systolic blood pressure 160, diastolic 115; a faint trace of albumin and a few granular and hyaline casts. There was no objective disturbance of the motor system. The strength of the right hand was equal to that of the left, but he could not use the right hand as dexterously as the left. There was no disturbance of station or gait. There was no disorder of the skin or of the superficial reflexes. Sensation, including deep sensibility and stereognostic sense, was intact.

The diagnosis of chronic nephritis—generalized arteriosclerosis and cerebral thrombosis—was made and appropriate treatment ordered. He was not seen again for six months, when he returned, the symptoms being very much worse including this time a tremor of the right upper extremity which developed about six weeks after his first visit. Latterly it had increased to such a degree that it made it difficult for him to use his right hand at all. He complained also of headache and vertigo, but the chief complaint, aside from tremor, was of burning pain in the right arm and shoulder. The movements on closer examination were seen to be somewhat complicated.

There was a slow, coarse, rhythmical tremor of the paralysis agitans type of the right hand and arm when at rest. When the arm was in motion, a jerky irregular tremor appeared, increasing in intensity as the member reached its goal. The cogwheel type of resistance to passive movements could occasionally be noticed and the primitive movements of the right hand were disturbed. In addition, these were distinctly athetoid in type. They consisted of a semiflexion at the elbow joint, slight extension of the wrist, followed by a slow, steady flexion, and extension of the fingers. These were not constant in their occurrence nor were they controllable. When the patient was lying at rest the right lower extremity was seen to display occasional involuntary jerkings, slow in character and arrhythmical. They were not synchronous with the movements of the right upper extremity. The facial expression was immobile, but the general attitude was one of mobility.

There was no discernible diminution of strength in the right side of the body. He walked with a distinct limp, but there was no motor paralysis nor indeed could a muscular hypertonus of the right side of the body be demonstrated. He could not, however, use the right upper or lower extremity dexterously. The tendon-jerks were quite normal, likewise the superficial reflexes. There was a big toe phenomenon and no digital reflex. General sensibility had not changed since the first examination. The systolic blood pressure was 150, and the urine contained albumin. Examination of the interior of the eyes gave no indication of the advanced nephritis.

Diagnosis.—An analysis of the objective signs of this case shows a tremor of the paralysis agitans type when the arm was at rest, an intentional tremor with the amplitude greatest when the focus was reached, occasional

athetoid movements and spasmus mobilis, immobility of the face and occasional passive resistance of the cog-wheel type, disturbance of the primitive movements of the hand, and a shuffling limp which in no way resembled a hemiplegic gait. These three varieties of involuntary movements occurring in the same individual are, as far as I know, rather unusual. Combinations of two sorts have been described. C. Vogt and Oppenheim have published cases of Parkinson tremor with choreo-athetoid movements, and an intentional character in paralysis agitans tremor has long been known. The latter has usually been described as differing from a true intentional tremor in that the amplitude is greatest about half way before the goal is reached. This certainly was not so in this case, for it behaved in nearly all respects as a true intentional tremor. It differed somewhat from the tremor of Wilson's disease, because during the excursion of the limb the rhythm was completely lost in the coarse, jerky, irregular character of the movement. It cannot be denied, however, that the presence of these three varieties of involuntary movements has some significance, and it may very well be that they are more closely related than was formerly thought to be the case. This is more than ever probable, if we accept Wilson's views on the pathogenesis of choreo-athetoid movements, namely, that they result from failure of the normal centripetal impulses which pass over the afferent cerebellorubrothalamicocortical tracts to the cortex. This view is in direct opposition to the older conception of a lesion producing irritation of, but not blocking, the pyramidal tracts, and certainly seems more reasonable. Wilson also explains the tremor as a disturbance of the efferent extrapyramidal pathway of the lenticulorubrospinal tracts, in which a destructive lesion of this pathway deprives the anterior

horn cells of the normal inhibiting influence of the corpus striatum. The anterior horn cell becomes weakened by this consistent flow of impulses, and the greater the innervation of the pyramids, the more violent the tremor becomes.

It becomes apparent, therefore, from perusal of the above, that the combination of involuntary movements found in this case may be explained by lesions involving the two pathways mentioned, especially if we consider that the primary focus in the lenticular nucleus extends towards the external nucleus of the thalamus. Lesions in this region causing a somewhat similar clinical picture have been described by Jelgersma, and it surely is not unreasonable to postulate a disturbance of the afferent and efferent pathways described by Wilson as well.

BRAIN INJURY RESULTING IN CROSSED PARALYSIS

C. BURNS CRAIG, M.D.

Traumatic disorders of the nervous system are of peculiar interest at this time because of efforts of various states, particularly New York, to establish by legislative act, just compensation for injured employees. The process of determining the degree to which the employer is responsible for the employee is still in progress. In the case presented, a man, 19 years of age, was employed as a clerk in a steel mill. On the 4th day of February, 1913, while working on the second floor, he was overcome by coke fumes from a furnace and fell through an opening, 30 feet, to the first floor. The greater part of the fall was borne by the left shoulder which struck a steel girder, the head striking a protruding bolt, laying open the scalp for a distance of six inches and carrying away some of the bone.

According to attendants he was unconscious for eleven hours. His first recollection began two days later when he discovered that the right side of the face was stiff and immobile and that he was absolutely powerless in the left arm and leg. After a period of two weeks he began to move the left thumb and then gradually the whole hand. After a period of six weeks he was able to walk, supporting himself by holding articles of furniture, dragging the left foot as he walked. From that time until the latter part of July, 1913, his improvement was ex-

ceedingly rapid so that he was able to walk in a manner almost normal and regained largely the use of the hand, though it was not so dexterous as the normal hand in performing fine movements. The patient is of the opinion that since July, 1913, there has been no improvement in either the arm, leg, or the right side of his face. The only other incident of interest in the history is that he suffered two convulsive seizures about one year after the accident. They were general, involving all extremities and facial muscles. He was not unconscious during them and there has been no recurrence.

Physical Examination.—Upon examination a large scar is evident extending from the right supra-orbital region upward across the forehead about to the coronal suture, then transversely across the scalp to the left frontal region. In the right supra-orbital region the end of the finger can be inserted into a small defect in the bone.

Examination of the cranial nerves shows the following disorders. Anosmia in the right nostril; defective sense of smell in the left to such a degree that strong odors, *e.g.,* turpentine, produce a sensation of odor but it cannot be identified. Vision is diminished in both eyes. This is due to a high degree of myopia which is greater in the right eye than in the left. The fundi are normal. The extrinsic muscles of the eyes are competent. There is myosis of the left pupil. Reaction to light and accommodation are prompt. There is a pronounced enophthalmos on the right side. There is a mild degree of hypesthesia over the entire right side of the face including the cornea. Strong flavors are not perceived on the right side of the tongue. The right side of the face is completely paretic, both the occipitofrontalis and the muscles below the orbit. Hearing is acute in both ears. Investigation of the auditory apparatus by Dr. Dench reveals an intact

labyrinth. The palate is symmetrically elevated. There is no symptom relative to the vagus nerve. The trapezii and the sternocleidomastoid muscles are symmetrically innervated. The tongue is protruded in the median line without deviation.

The patient's gait is almost normal, but there is slight lagging in the left lower extremity which would not ordinarily be noticed. His station is secure. The upper extremity exhibits a slight retardation in attempts at rapid movement, such as punching a bag, but the strength is good, as is also the grasp of the hand. Adiadochokinesis is present in the left arm. The knee-jerks are present, the left being slightly greater than the right. This is also true of the ankle-jerks. Babinski sign is present on the left, but there is no clonus. The left abdominal and epigastric reflexes are diminished. Hoffman sign is present in the left hand.

There is a mild degree of hypesthesia to all forms of stimulation over the left arm and leg and the left half of the trunk.

X-ray examination showed no depressed bone.

Diagnosis.—To summarize, the patient presented a partial paralysis of the first, fifth and seventh cranial nerves of the right side, and disturbance of the intracranial sympathetic system on the same side, and a lesion involving both the motor and sensory fibers to the left half of the trunk, the left arm and leg.

In order to explain satisfactorily these findings it is necessary to suppose that there was besides the evident lesion to the skull, a fracture of the base which involved the right first, fifth, and seventh and the sympathetic, and that a meningitic process supervened involving these nerves. The motor and sensory disturbance to the left

side of the body must be explained by a cortical lesion on the right hemisphere.

Subsequent History.—After three months' continuous supervision and treatment at The Neurological Institute, the patient showed distinct improvement in both the ability voluntarily to move the right side of the face, and in the use of the left upper extremity. The course of therapy was vigorous and without respite. The patient was made to practice various grimaces, using chiefly the right side of the face at repeated intervals daily. He was supervised in daily exercises, calculated to educate the dexterity of the left upper extremity, such as throwing a ball, using a foil, typewriting with his left hand, or practicing the piano movements with his fingers. This course of treatment was supplemented with the usual tonic measures, such as baths, massage and electricity. The result has amply repaid in the improved use of the disordered muscles.

THE MENTAL SYMPTOMS OF CONSTITUTIONAL INFERIORITY DUE TO HEREDITARY SYPHILIS WITH NEGATIVE WASSERMANN REACTIONS AND THE EXISTENCE OF THE ARGYLL-ROBERTSON SIGN

WALTER CLARK HAUPT, M.D.

The patient, an Italian girl, 17 years of age, came to the dispensary October 5, 1916, complaining of insomnia and of seeing at night all kinds of animals on the floor and serpents on the ceiling. Her mother added that she talked peculiarly and irrationally at times, that occasionally she did not seem to know the members of her family and that she was generally depressed, crying a great deal and expressing hatred for everything. A few days before entrance she insisted on leaving the house at four o'clock in the morning with her mother, because she could not stay there any longer. The mother had eleven children, seven of which are living, the patient being the youngest. The father is alive and well at 71 years of age. One brother at the age of 20 years spent four weeks in a sanitarium in Italy. The mother states that while her daughter had never had any acute mental symptoms until about three months ago, she had been considered peculiar since coming to this country four years ago. She had had no serious illness; she has never been friendly and she has had little application, being unable to continue any kind of work that she may have

started. The girl herself has some appreciation of this fact. This condition has been noticeable chiefly during the past three months; up to that time she apparently worked satisfactorily and intelligently, earning as much as seven dollars a week. She cannot recall when she stopped work. The mother, as well as her brother and a friend, report that her acute mental symptoms date from the powder explosion near Bedloe's Island, in July, 1916, which frightened her severely. She went to Liberty, New York, and became sleepless the following week, anxious, afraid of an indefinite something.

After admission to the hospital for further examination, she spent a very restless night, crying, getting in and out of bed and manifesting symptoms of profound agitation. She complained to the nurse that she felt as though there were worms inside of her body, that she could feel them moving around, particularly in her abdomen. She felt as though everything had stopped inside of her body. In the morning she got up and dressed and walked around the room, crying, complaining of the same sensations.

On physical examination she was found to be a well-built, well-nourished girl, whose appearance did not indicate mental disorder. The only positive findings were A.-R. pupils. The right pupil was pin-point in size, the left moderately dilated, and both irregular in outline. The left was oval in shape, the long axis being horizontal. Neither responded to light and very sluggishly to accommodation. Her serology on two occasions was negative throughout. In the colloidal gold test there was no discoloration except in tube five in which there was complete precipitation. (It is interesting to note that Wassermann tests of the serum of the patient's mother and brother were positive.)

Apparently the girl did not comprehend questions at times, even when addressed to her in Italian. She was oriented as to time, place and person, though there were lapses during which she gave no answer to any questions pertaining to these topics. She was given a pencil and paper and asked to write when she came to the hospital, what had been done for her here, whom she saw, and in a general way to give a brief description of her few days' stay, but she did not produce a word, adding, "That is the trouble; I can't think, nothing comes into my head; when I try to think my mind gets empty." After repeated urging, she was induced to write her name and then the following in Italian: "The first time that I came to this hospital was October 5th. I came to have a good examination by the doctor."

The report on her reactions to the Binet-Simon intelligence test was as follows: "The patient's mental condition is backward. She had difficulty in understanding English, also in thinking and expressing herself in English, yet on the whole, the difficulty seemed to be one of thinking rather than of language. She has learned to write well except for occasional flourishes and to spell fairly correctly. She knew the date, though she was easily confused by unfamiliar language; she worked slowly with long waits; effort seemed not distasteful, but she lacked interest, as if dreaming. She whispered to herself; her motor performances seemed steady, though slow, and not vigorous. Her reply to geometrical forms was poor, that to syllables likewise. Her drawing was occasionally fairly good; it was sometimes a little fanciful. She marked over her lines often, and she hesitated in her work, though she did not seem really oppositional. She lacked courage and confidence, and seemed somewhat distressed; she was suggestible; she was sentimental and apparently

enjoyed lofty forms of expression; she was asked to write poetry for her word list and wrote twelve lines of Barbara Fritchie well, *i.e.*, with only an occasional senseless expression. She wrote only three words in twelve minutes for her word list. She thought with only the most meager supply of language."

There was no decided memory defect. She was able to tell when she came to this country, how old she was at the time, and to relate events that transpired during her stay in the hospital. She was depressed and hated everything, she often repeated. On specific questioning she denied any visual or auditory hallucinations. She had no ideas of reference, nor did she express delusions of persecution or of grandeur. In general her mental status impressed the observer as an episode in a constitutional inferior.

Diagnosis.—Argyll-Robertson pupils are universally considered to be a positive sign of syphilis of the nervous system. In a statistical summary Siemerling made on the basis of 1639 cases of A.-R. pupils, 94.7 per cent occurred in persons suffering from general paresis, tabes or cerebrospinal syphilis. A.-R. pupils in a patient with mental symptoms point to general paresis. In this patient the mental symptoms are not those common in paresis, however. In view of this and the negative laboratory reports it is unjustifiable to make this diagnosis. In spite of this when we take into consideration the positive Wassermann of the mother's and brother's sera, we may assume that the patient has hereditary syphilis. If a definite diagnosis had to be made at the present time it would seem most probable that we are confronted with an episodic hallucinatory state, the result of hereditary syphilis. It is quite legitimate to consider the mental manifestations the expression of a shock psychosis.

Subsequent History.—This patient made a complete recovery. She became cheerful, affable, docile, industrious, responsive, and she has remained so now nearly a year. Then she had an episode paralleling the above. No antisyphilitic treatment was administered. The brother's serum remained positive despite active mercurial and salvarsan treatment.

A CASE OF CEREBRAL SYPHILIS

JOSEPH COLLINS, M.D.

The clinical picture of brain syphilis is so variable and often so extraordinary that we should record every case in order that the task of him who is destined to be the future Fournier may be easier. The case here presented is one of a class of which we have had four examples during the past year. In a general way it may be said to present the picture of an overwhelming intoxication.

The patient, twenty-six years old, was brought by his mother, who said that he had become entirely unlike himself physically, mentally, and emotionally; and all within the past few months. The history obtained from her and his physician is as follows:

When he was 20 years old, he had a chancre. It lasted for six months. He did not go to a doctor nor did he himself apply any remedies. So far as he recalls now there were none of the so-called secondary manifestations of syphilis. Six months ago he took a young woman to her physician because of some trouble with her throat. The doctor diagnosticated syphilis. The young man then announced that he had contracted a secret marriage with her a short time previously. His blood was taken and the Wassermann was found to be positive. The physician put him under treatment with hypodermic injections of salicylate of mercury. The first was given on May 1st. He gave one about every five or six days and altogether he had about twenty-five treatments. At the beginning

of July he developed a right-sided facial paralysis, all branches of the seventh nerve being affected. This paralysis lasted several weeks, then gradually began to disappear. The evidence of its existence was still pronounced when he was first examined here. Apparently there were no very considerable symptoms attending this facial paralysis. The young man went to his business, that of clerk in a drapery store, until the 25th of September. On the morning of that day he left his house as usual and about fifteen minutes after leaving he was brought in by a policeman and a stranger, who said they found him lying in the street. He related that he was seized with dizziness and had fallen. After being put to bed he complained of nausea, headache, and dizziness, and vomited several times during the next three or four days. During the first week after the onset of this attack he was stuporous and disinclined to converse or take much interest in anything. Gradually his condition returned to that of approximately normal, and he went to his work again. A week or two ago he was requested to go home and remain there until he got well. He himself, apparently, did not realize that there was any great change in his make-up. He was merely "run-down." His mother, however, said that he was quite unlike himself, did strange things, drank when lying down, ate like an animal, stuffed his mouth full and kept on stuffing it. He did not fully realize the things he did, and he made false statements.

Physical Examination.—On physical examination he was found to be an emaciated, pale, sickly-looking young man, weighing 100 lbs. While being examined, clammy perspiration broke out on his legs. His station and gait were normal, but insecure. There was a decided right facial weakness. There was no tremor, spasticity, or in-

coördination. His patellar and Achilles jerks were both exaggerated, the right more so than the left. On the right side there was a patellar and ankle clonus. On plantar stimulation there occurred a fan-like extension of all toes. Babinski sign is present on both sides, more marked on the right. Abdominal, epigastric, cremasteric reflexes as well as those of the upper extremities were present. The eyes were normal except for inequality of the pupils. The right was larger than the left. There were no disturbances of sensation. His speech while laconic, inebrious, and at times indistinct, showed no definite stumbling or hesitation. When examined, he resented scratching on the sole of the foot, complaining that it hurt him. When he was pulled out on the side of the bed and made to get up, he stood and walked about the room as though he were half asleep, or semi-intoxicated. There was no trace of hemiplegia, however. His serology was positive throughout, with 160 cells in the cerebrospinal fluid. The colloidal gold showed a syphilitic curve, not a paretic one.

He was in a semistuporous condition and difficult to arouse, and when aroused, did not remain so, but lapsed back into a position as if he were overwhelmed by sleep. His answers to questions were characterized by triviality and casuality. His attitude when aroused was one of genial bonhomie; he smiled readily in a supercilious manner. When asked how he felt, he replied in a stupid, inebrious way, "middling."

"Are you married?"

"No."

"Who is this girl your mother says you married?"

With considerable snap he retorted, "So you insist on bringing her into it?" Immediately afterward he relapsed into a stuporous state in which he remained with his eyes closed.

After a while he was asked, "Are you asleep?"

His answer was "No."

"Why do you close your eyes?"

"Just for fun."

He could not give the date, insisting that it was April and summer time.

"What are you doing here in the hospital?"

"Search me."

"Who has been elected president?"

"Wilson."

"Whom did you vote for?"

"My vote counts for naught."

"How is that?"

"It was not counted."

"Why not?"

"I did not vote."

"Are you a Republican?"

"Yes."

"What is the difference between a Republican and a Democrat?"

"When you come down to platforms there is very little difference."

"Who was president before Wilson?" No answer. "Who was the first president?"

"Washington."

"Other presidents?"

"Lincoln."

"When did he live?"

"Time of the Revolution."

"Who was president at the time of the Civil War?"

"Lincoln."

"When was the Revolution?"

"Early."

"Signing of the Declaration of Independence?"
"1776."

He could not be made to see the discrepancies in his answers. Falsification of memory could not be demonstrated. He did not realize that he was sick and could not account for his stay in the hospital, insisting that he was merely run-down. His memory seemed to be better for remote than for recent events although it was defective even for the former. His mental reactions in short were the following: a rather sudden alteration in personality, showing itself in neglect of good form at home as well as in business, indicating the liberation of the customary inhibition governing good conduct; a semistuporous condition. Even when aroused therefrom he was apathetic and showed distinct clouding of consciousness, triviality and a certain flightiness in his answers.

Diagnosis.—As regards the pathology in this case, it seems most probable that the lesions are to be found chiefly in the meninges. The patient's symptoms are very much like those found in cerebromeningeal edema. Indeed it is not unlikely that the lesion constitutes an acute meningocerebritis. He was started on vigorous salvarsan and mercurial therapy, and soon showed signs of improvement both serologically and clinically.

Subsequent History.—This patient was presented to the Conference two weeks later, *i.e.,* on November 29th, in order to demonstrate the remarkable improvement resulting from the one intravenous (.3 gm.) and two intraspinal (.3 mg.) salvarsan injections, as well as daily inunctions and bichlorid of mercury injections. The outstanding features of the improvement in this case were: the return of the patient from a semistuporous state to complete consciousness, orientation, and an almost normal attitude and behavior. He was up and about, careful

as regards his appearance, and participated in the roof exercises. His memory, from which the happenings of the past three weeks appeared to be completely blotted out, proved quite good for events preceding the acute onset of his illness and those of the past few days.

Laboratory Findings.—The third feature of his improvement was the change which had taken place in his serology. The latter was positive throughout, with 160 cells, on entrance. At this time the serum and cerebrospinal fluid Wassermann were both negative, the globulin was weakly positive and the cell count was reduced to 11.

This patient was kept under treatment for six months during which time he received twenty intravenous injections of salvarsan, twelve intraspinous and a great deal of mercurial medication. He was examined a year from the date on which he was first seen. He was considered to have made a complete recovery and it was very difficult to say in just what way he was not fully well. He seemed to be intelligent, reasonable, tractable, interested, capable of earning a livelihood and going amongst others without exciting adverse estimate but still his mother maintains he is not quite well, not quite like his former self. One would have the same difficulty in describing the peach from which a bit of its bloom had been removed. There are no somatic or laboratory indications of syphilis but he was tarred with the brush that leaves an indelible mark.

CEREBROSPINAL SYPHILIS WITH DISPLAY OF SYMPTOMS SIMULATING POLIOMYELITIS

JOSEPH COLLINS, M.D.

It has been frequently stated here that cerebrospinal syphilis has a very great variety of clinical displays and that those displays have not heretofore been fully nor adequately described. They rarely simulate poliomyelitis. In the case herewith presented the symptoms were quite parallel to those not infrequently seen in the epidemic of poliomyelitis that prevailed in 1916. When the symptoms of poliomyelitis develop abruptly in an individual who has had syphilis—whose blood and cerebrospinal fluid reveal evidences of syphilis—and when such symptoms disappear under antisyphilitic medication, it may properly be assumed that the disorder is syphilitic poliomyelitis.

The patient, 34 years old, employed as a polisher of umbrella handles, was in good health until three or four days before he came to the clinic, December 4, 1916; then there developed inability to use the right hand owing to the weakness of the wrist. Two days later the right side of the face became paralyzed. It was for the weakness of the right hand and paralysis of the right side of the face that he sought relief. He admitted, however, on repeated questioning, that three weeks before these symptoms developed he had some fatigue in the right hand, but as it did not interfere with his work he paid no attention to it.

He used very little lead paint in his work, but a great deal of varnish and alcohol. He never had any complaint that suggested lead poisoning.

Seven years ago, that is, when 27 years old, he became infected with syphilis. The evidences of infection were a typical local lesion, erythema, and alopecia. He was treated with mercury, administered hypodermatically. He thinks he took about thirty injections, and, as they were given several days apart, it is fair to assume that a non-soluble salt was used. His wife has had no manifestations of syphilis since that time; she has borne two children and is now pregnant with the third.

Physical Examination.—The patient displayed right facial palsy of the peripheral type. The frontalis, however, was only slightly affected. The corrugator and orbicularis palpebrarum were more definitely involved, and the patient was unable to move the lower part of the face. He could close the right eye, but could not hold it as tightly closed as the left.

The upper right extremity was affected *in toto,* but there were certain muscles which were more markedly affected than others, especially the extensors of the hand and wrist. The paralysis was of the flaccid type and tended downward, that is, from shoulder to the hand. There was no atrophy and no evidence of fibrillation.

The tendon-jerks of the lower extremities were lively, but not particularly exaggerated, and the cutaneous reflexes normal. The reflexes of the right upper extremity were present and lively; the biceps and triceps jerks decidedly so; the tendon-jerks of the left side quite normal.

There were no sensory disturbances, superficial or deep, and the other special senses seemed quite normal. All the muscles of the upper extremities and face re-

sponded to both faradism and galvanism. In the forearm flexors on the right there was slight reduction to the faradic response as compared with that on the left. The same was present to a lesser degree in the muscles of the right hand.

To faradism the muscles of the right face showed a definite reduction in both promptness and completeness of response. To galvanism no changes were demonstrable. Neither formula changes nor reaction of degeneration were present.

Examination of the blood and cerebrospinal fluid showed that all the reactions indicating syphilis were strongly positive; there was excess of globulin and the fluid contained 85 cells to the centimeter. There could be no doubt about the causative factor of his illness. He received a dose of salvarsan intravenously and vigorous mercurial therapy was initiated. On reëxamination, one week later, several changes were noted in his objective symptoms, the most striking of which were: (1) marked increase in muscular power of the extensors and flexors both of the wrist and the fingers, and, to some extent, of the forearm; (2) slight atrophy which had become evident in the thenar and hypothenar eminences and interossei of the right hand; (3) hyperactivity of the reflexes as well as increased myotatic irritability of the upper extremity, including the shoulder; exaggerated reflexes of the right leg as compared to the left; inexhaustible clonus which had developed at the right ankle; and an inconstant Babinski big toe phenomenon on the right side.

Diagnosis.—We are dealing, then, with a syphilitic patient who first presented himself with a flaccid paralysis of the right arm and a facial palsy of sudden onset and who within a week after antiluetic treatment partially

regained the muscular power both of his arm and face, while developing simultaneously slight atrophy of the small muscles of the affected hand and symptoms of pyramidal tract irritation. That the patient's symptoms must be ascribed to spirochetal infection there is no doubt. It remains only to decide whether we are confronted here with a faciobrachial palsy of cortical origin, a meningoradiculitis or a poliomyelitis. Against the probability of a cortical lesion the following factors speak: First, the onset of the patient's paralysis. As the face and arm area are supplied by the same vessel, one would expect a simultaneous involvement of the face and arm, whereas in this patient the arm became affected several days, possibly weeks, previous to the face. Second, the total absence of any mental symptoms. Third, the segmental involvement of the sixth, seventh and eighth cervical and first dorsal nerves, which is very uncommon in a cortical lesion. Fourth, the typical symptoms of a lower motor neurone lesion which the patient later displayed and the subsequent slight trophic changes. Fifth, the type of the patient's facial palsy which was peripheral, the upper facial muscles being less affected than the lower.

When we come to consider poliomyelitis and meningoradiculitis on a luetic basis, we can explain the patient's symptom-complex by assuming a lesion involving the lower cervical enlargement and a similar process in the middle fossa of the skull. We know from clinical experience and post-mortem examinations that such remotely separate foci of syphilitic diseases are by no means uncommon.

It would be most plausible to assume that the patient's symptom-complex was brought about by an initial meningeal process which, traveling inward along the roots

and blood vessels, affected the cells of the anterior horn and, extending along the side of the cord, caused irritation of the pyramidal tract.

The complicated symptomatology of the patient precludes an arbitrary differentiation between a poliomyelitis and a meningoradiculitis.

Subsequent History.—The patient made a complete recovery in less than six months.

JACKSONIAN EPILEPSY OF LUETIC ORIGIN

J. L. JOUGHIN, M.D.

An Irishman of the laboring class, aged 43, complained of generalized convulsive seizures with loss of consciousness, and of localized convulsive seizures affecting the left side of the body, not accompanied by loss of consciousness; the former seizures had troubled him for six years, the latter for two years.

When he was 34 years old, he had a chancre and received at intervals, for some years after, what is ordinarily called mixed treatment. In addition, he had received three intravenous injections of salvarsan. For three years after contraction of syphilis his health remained unimpaired. At the expiration of this period the first generalized convulsive attack occurred accompanied by biting of the tongue, involuntary micturition and unconsciousness. These seizures, at first rare, gradually became more frequent and, so far as can be ascertained, were identical with the "grand mal" attack of so-called idiopathic epilepsy. There was at this time no preponderance of the seizure in the left half of the body. With the onset of the localized attacks the generalized type of convulsion became much more rare.

The patient had headaches at rare intervals, but never of a severe type and never nocturnal. There was no history of diplopia or cranial nerve palsy. At no time was there any real aphasia but after a severe convulsion there was always transitory dysarthria or anarthria. There were never any monoplegic or hemiplegic seizures.

Polyuria existed for some months before the onset of the convulsions, the patient claiming that he urinated at frequent intervals and in large quantities. There was no complaint of vertigo or of vomiting. His eyesight remained unimpaired. Repeated interviews revealed no evidence of mental deterioration, his orientation, memory and attention being unaffected.

Physical Examination.—The cardiovascular findings were negative. There were neither subjective nor objective changes in the sensory sphere. When the motility of the left hand was sufficient for accurate palpation of the test objects, no true astereognosis existed—an apparent astereognosis found being evidently due to motor deficiency. His tendon-jerks were present on both sides, but increased on the left; on neither side was a true clonus obtainable. The abdominal, epigastric and plantar reflexes were normal and equal. The cremasteric, present on both sides, was less active on the left. The examination of the eyes revealed nothing abnormal. The peripheral nerves were intact.

Display of Symptoms.—During the examination at intervals of 10-15 minutes he had typical Jacksonian attacks which may be thus described. They began by a gradual hyperextension and separation of the fingers of the left hand and arm. The patient complained that he was afraid that his fingers would break at the knuckles and forcibly flexed them with the other hand. The arm became semiflexed and rotated outward and the member became fixed in this position by a tonic spasm, the hand being gradually raised above the level of the head. As the clonic phase occurred the arm lowered to its original position, movements of extension and flexion occurred in the arm and fingers, and finally complete relaxation supervened. When the leg was affected it became

semiflexed, the foot extended, somewhat inverted and the big toe strongly dorsiflexed. The facial involvement was indicated by the face deviating to the left; the left angle of the mouth was drawn upward causing the mouth to gape. The eye was closed and there were repeated rapid contractions of the orbicularis and occipitofrontalis muscles. In the more severe convulsions an intense cyanosis was manifest. In all attacks of any severity there occurred a marked conjugate deviation of the head and eyes to the left, which the patient could not voluntarily inhibit. This he attempted to overcome by placing the right hand on the chin and endeavoring forcibly to pull the head to the right. There was no nystagmus. The intensity of the attacks could be often ameliorated and not rarely they could be entirely suppressed by his firmly constricting the left wrist with his right hand, or better still, having some one else do it for him.

His description of the aura was unsatisfactory. There appeared to be no definite sensory aura, but he complained of a "shock" felt in the left side of the chest which sometimes preceded the fits. On several occasions he arose and rotated the body several times to the left before the actual convulsion occurred. The attacks varied greatly in severity and always began in the left hand.

During the two days subsequent to admission to the hospital, the attacks increased in severity and number, 40 to 50 occurring within 24 hours. Gradually there developed a flaccid left hemiplegia involving the face, the facial involvement being of the upper motor neurone type. The abdominal and cremasteric reflexes became sluggish on the left, and the plantar response on that side disappeared entirely. On the right no alteration was observed. These findings taken in conjunction

with the character of the Jacksonian fits warranted us in postulating the existence of a cortical lesion. With this hemiplegia existed an anarthria so complete that the patient could not utter a word.

This severe series of convulsions also markedly affected his mental state which deteriorated rapidly, so that in a short time he showed pronounced evidence of mental confusion and complete disorientation. This mental confusion and disorientation soon disappeared, but he developed on coming out of the confusional state a peculiar paranoid symptomatology with delusions of reference apparently based upon auditory hallucinations or illusions. The reason he gave for the persecution to which he was subjected by the other patients and one of the doctors was that he protested when a young boy in the room was teased by other inmates of the ward. They threatened to punish him for this, saying, "We'll make him have another fit." "We'll beat him up when he gets out," etc. A hose was dragged into the room during the night to squirt on him. The doctor tried to poison him by the injections and he would take no more. Patients on the roof insulted him and threatened to hit him; they were always talking about him. Having asked the examining physician for one of his cards, immediately after receiving it some one in the next room said, "Don't take it, it is all a bluff." (As a matter of fact dictation regarding another case was taking place in the next room but no words of this character were uttered. Auditory illusion?) Several months later a number of these beliefs still remained fixed in the patient's mind.

The examination of the spinal fluid and serum revealed nothing abnormal, and other laboratory examinations were also negative. The treatment given was at first eliminative and sedative. This was followed by antispecific

therapy consisting of one half-dose of salvarsan and two half-doses of neosalvarsan. After the administration of the first dose all convulsions ceased; his motor power was continually augmented and his mental state showed rapid improvement. Under further antisyphilitic treatment he made what seemed to be a complete recovery.

Two weeks after the administration of the salvarsan the tendon reflexes were lively but equal and there existed neither patellar nor ankle clonus. The epigastrics were equal; the left abdominal and left cremasteric were very sluggish, especially the latter. Plantar flexion was difficult to elicit on both sides but more so on the left. There was nothing even remotely suggestive of a Babinski.

The pupillary reaction was somewhat altered. The left pupil was normal as it was at the time of admission, but the right was now decidedly larger than the left; it was a little irregular and reacted to light sluggishly. The hemiparalysis had been replaced by a hemiparesis which was retrogressing. The speech disturbance was no longer anarthric but dysarthric, and when he became excited and test phrases were employed it was suggestive of the speech of paresis. This as well as other types of speech disturbance are not uncommonly met with in cerebral lues involving the cortex.

A neoplasm may be ruled out, owing to the absence of all symptoms of increased intracranial pressure commonly accompanying such growths, which we might assuredly expect to have manifested themselves in a new formation growing for more than two years. This naturally is not meant to exclude gumma formation in the right frontal lobe, an integral part of any existing gummatous meningitis.

Diagnosis.—The long course of the disease, the lack of the peculiar somatic signs, of psychic deterioration, and the absence of positive findings in either the serum or the cerebrospinal fluid may be considered to exclude general paresis.

In spite of the absence of positive laboratory reports, the correct diagnosis here is in all probability lues cerebralis. The definite history of infection, the onset of the convulsions at a relatively advanced age, the favorable result of the specific therapy and the absence of any other cause to which the symptoms may be attributed, appear amply to justify this assertion.

The localization and pathology of the lesion is, as it is so often, in even nonaberrant types of cerebral syphilis, very largely a matter of individual opinion.

That the involvement of the base was of slight degree may be inferred from the absence of such symptoms as choked disc, cranial nerve palsies, etc., peculiar to lesions of that locality. The history was equally free from symptoms, with the exception of the transient polyuria, which would incline us to accept this as the principal localization of the syphilitic process. To decide whether we were dealing with a localized gummatous meningo-encephalitis of the convexity or with a specific arteritis with cortical areas of softening is much more difficult, as both these types of lues produce a symptomatology often nearly identical. Possibly both conditions were present. That the symptoms, however, were due to cortical lesions, no matter in what manner produced, appears certain.

The speech disturbance, the alteration of the cutaneous reflexes, and the peculiar character of the convulsions so clearly indicating a lesion affecting the posterior extremity of the second right frontal convolution and the

hand and arm centers of the right ascending frontal convolution, all force us to accept such an interpretation.

There is no attempt here to urge exclusively the existence of any particular type or localization of the pathological process, the well known tendency of cerebral syphilis to invade the various anatomical structures of the brain and its membranes, renders it very probable that more than one variety of lesion existed.

CEREBRAL SYPHILIS WITH UNHERALDED FATAL TERMINATION

JOSEPH COLLINS, M.D.

Sudden death from syphilis is not altogether uncommon. It is due usually to abrupt occlusion of a blood-vessel in the pontobulbar region incidental to endarteritis or to cardiac thrombosis. The case that I report was not one of sudden death but of unexpected and unheralded death.

On November 16th, I was consulted by a man 38 years old, who sought relief for excruciating pain which he had had for about a week. He gave a history of having been infected with syphilis when he was 33 years old. He was treated in the customary way with mercury and iodids and had never taken salvarsan. He had been quite well until about the middle of August, when he began to be irritable and nervous. A few weeks later he noticed weakness of the fingers of the right hand. But it was not until November first that he gave heed to his symptoms, when his employers complained that his work, as foreman cutter in a manufactory of clothes, was not satisfactory and that he was making costly mistakes. On November first, he went to Atlantic City for a week and then returned to work. Soon he began to complain of pain in the head, at first inconstant, but increasing day by day both in severity and constancy. He kept at work until November 16th. The pain was described as stabbing and darting, as if a lot of knives were being thrust

through the back of the head into the brain. It was difficult to elicit any further story from him, the pain was so great that it over-shadowed everything else. He was sent immediately to the Neurological Institute, New York City, and a lumbar puncture done.

Laboratory Findings.—The report the following morning on the spinal fluid and on the blood was:

Blood Serum Wassermann, Weakly	+
Cerebrospinal Fluid Wassermann	+
Globulin	+
Fehling's	+
Cells	117

(115 lymphocytes, 2 polymorphs.). Colloidal gold test showed a negative paretic curve.

Physical Examination.—The results of physical examination were meager; the tendon-jerks were lively on the right side of the body, rather sluggish on the left. The superficial reflexes were normal. There was no speech disturbance, no tremor, no rigidity. The pupils responded promptly to light and the optic discs were normal. In brief, there were no somatic manifestations of syphilis. The diagnosis was made from the history, the pain and the laboratory report. It was impossible to make a satisfactory mental examination. Whether he was stupid from pain or from the lesion could not be satisfactorily established. On the 17th he was given 0.3 mgm. salvarsan (salvarsanized serum) intraspinously. The pain in the head was kept within toleration by small amounts of aspirin and phenacetin. The next day he felt better and gave a coherent account of his illness, which did not differ materially from what has been related. In the evening the pain returned again and he became quite excited. On the 19th he again had a fairly comfortable day, but a

bad night, and the following morning he seemed confused and refractory. He did not want to stay in bed or to keep his clothes on; he bit the thermometer when it was put in his mouth, etc. During the day he appeared to be drowsy and very confused. He was given ⅓ grain bichlorid of mercury and iodid of potassium in twenty grain doses internally. The following morning he had an involuntary discharge from the bowels after administration of magnesium sulphate. During the day he complained of headache and was stuporous. After being aroused, he would answer laconically and immediately close his eyes and sleep. Again he would rouse abruptly, shrieking with pain. Pulse, temperature and blood pressure were quite normal. The only abnormality of the urine was its alkalinity.

When I went into the room he was sitting on the edge of the bed with head in his hands and an expression of agony and of exhaustion. I asked him if he had a headache and he replied, "Terrible." "How long have you had it?" "All the afternoon." "But it is not afternoon now, it is morning." I showed him my watch; it indicated 11.25, and he said that it was 12.25. Then he kept repeating, "It was afternoon." "When was your wife here?" "Yesterday afternoon, this afternoon, this afternoon, this morning, this afternoon." "Where did you see her?" "This afternoon." "Yes, but where did you see her?" "This afternoon." Despite this apparent inability to reply to a question, the moment I said to him "Push up a little further in the bed," he did it with alacrity. When asked when he saw me first, he could give no account of it whatsoever. He was like a person in some stuporous state. There was no evidence of any stiffness of the neck or rigidity of any part of the body.

Physical examination in brief showed no paralysis.

The exaggerated tendon jerks were more pronounced on the right side of the body (not pathologically altered but lively knee, ankle, elbow jerks). On the left side of the body the tendon jerks were slightly below normal, and the remarkable feature was that he had a definite left Babinski; on the right side there was an approximation to a Babinski which might be construed to be a defense movement, save that the big toe remained in extension. The superficial reflexes were all present. There was no disorder of the motor system. Examination with the ophthalmoscope showed beginning papilledema of both discs, the elevation being 1 D. Lumbar puncture was done at this time and 15 c.c. of a clear fluid which escaped under considerable pressure was withdrawn. Examination of this fluid showed practically the same condition as before, only the number of cells was now 38. He was incontinent, but whether this was incident to his mental state or a genuine incontinence, it was impossible to say.

In the afternoon he went into a state of collapse. The pulse became feeble and intermittent, the extremities cold, the body bathed in cold perspiration, and his whole expression was that of agony. On listening to the heart it was found that many of the systoles were not represented in the pulse. There was no dyspnea. He was given atropin hypodermatically, and in ten minutes the attack terminated, but two hours later he had another attack which lasted somewhat longer. On this occasion he was given morphin, followed by digipuratum. Two hours later he had a third attack in which he succumbed, the immediate antecedent of dissolution being dyspnea. The pulse continued to beat nearly five minutes after respiration had ceased.

Diagnosis.—We were not permitted to do an autopsy;

therefore any opinion concerning the cause of the patient's death must rest on conjecture. The patient's symptoms and the information obtained from examination of the blood serum and cerebrospinal fluid suggested that the disease was a syphilitic pachymeningitis. It progressed with great rapidity although he had had some symptoms for approximately three months. He and his family limited the time of his illness to the month of November. Assuming that the lesion was a syphilitic meningitis it is difficult to reconcile the mode of dissolution with the course of that disease. It is barely possible that invasion of the spirochetes was not confined to the meninges, but that he had an acute parenchymatous syphilis of the brain and that the phenomena of dissolution were the expression of medullary invasion. It seems to me much more likely, however, that they must be attributed to the Herxheimer reaction. Were it not for the fact that the symptoms developed such a long time after the administration of salvarsan (72 hours) I should have very little doubt about it. My experience has been that the Herxheimer reaction occurs within 48 hours after the administration of salvarsan. I do not construe the intensification of the patient's symptoms which we occasionally see after salvarsan administration to be evidences of the Herxheimer reaction, although it is quite possible that in some instances that is the explanation.

MONOCULAR OPTIC NEURITIS AND PARTIAL BLINDNESS FROM DISEASE OF THE ACCESSORY NASAL SINUSES WITH OPERATION AND RECOVERY

J. L. JOUGHIN, M.D.

A young Jewish girl, aged thirteen, came to the Neurological Institute, New York City, May 17, 1917, complaining of complete loss of vision in the left eye.

Her history was as follows: For many years she had been troubled with recurrent attacks of acute coryza occurring principally during the winter months. On one occasion she consulted a rhinologist who informed her that some bones should be removed from her nose, but she never had this done. In December, 1916, one of these attacks to which she was accustomed occurred, which was accompanied by a phenomenon never previously experienced: a feeling of dizziness lasting for a few seconds and occurring many times daily. As the coryza got better these attacks decreased and ultimately disappeared after one month.

She then remained quite well until April, when she developed a severe infection, probably febrile, and accompanied by much coryza and general muscular tenderness. This cleared up after ten days. Shortly afterward she vomited once, but she had no cephalic pain except that outlined above. There was no history of aural discharge, but once when a young child she had a right-sided earache lasting one week. During the first week of May she

complained of disagreeable subjective visual sensations affecting the left eye, bluish balls of light floating before her, the appearance of which was especially induced by light pressure on the eyeball. Vision began almost immediately to diminish, and it decreased so rapidly that when she presented herself at the clinic on May 17th she could barely count fingers held 18 to 24 inches before the left eye.

Spontaneous pain in the eye was first experienced about one week before we saw her; and it occurred on looking down and to a less extent on looking up or in either direction laterally, and the same symptoms could be elicited by pressure on the eyeball. The spontaneous sensibility had then (May 18th) disappeared, but the tenderness on pressure persisted.

Summary and Chronology of Symptoms.—First: Recurrent attacks of acute coryza for many years. In December, 1916, an attack accompanied by transitory vertigo and lasting until the middle of January, 1917. Second: An acute febrile infection (grippe?) in April, 1917, accompanied by coryza. Recovery. Third: Subjective visual disturbances and rapid failure of vision beginning about May 1, 1917. Fourth: Spontaneous and induced pain in the left eye for the last week.

Physical Examination.—The general medical examination was quite negative. The right palpebral fissure was greater than the left. The pupils were equal, dilated and reacted well to light and accommodation, including the consensual reaction. There was no diplopia, no nystagmus. Vision was normal in the right eye; in the left she counted fingers only with the greatest difficulty. The right fundus was normal, but the left showed a high degree of optic neuritis, numerous small exudates and a few capillary hemorrhages. Vision 18/200.

Diagnosis.—The case was interpreted as a sinusitis involving the left sphenoidal or left posterior ethmoidal sinuses, that is to say, either one or both might be affected. The history of repeated attacks of coryza and the presumption that there existed some nasal abnormality, the absence of all symptoms regarded as indicative of increased intracranial pressure, the ocular tenderness and the monocular papillitis and failure of vision, all seemed to indicate that such was the correct diagnosis.

She was sent to Dr. John E. MacKenty of the Manhattan Eye and Ear Hospital for operation, who reported as follows:

Nasal Examination Prior to Operation.—The X-ray gave no assistance in making a diagnosis. The sinuses all seemed to be clear. There was no nasal discharge. The mucous membrane covering the ethmoid labyrinth was slightly congested, darker than normal, but there was no evidence of actual disease in the labyrinth.

On May 24th operation was done externally, the ethmoid labyrinth was removed and the exterior wall of the sphenoid entirely taken away. The mucous membrane lining of the ethmoids was thicker than normal. The mucous membrane lining of the sphenoid was normal. On the external wall of the sphenoid were several small excrescences, which may have been due to bone inflammation.

Subsequent History.—The eye examinations made by Dr. E. S. Thomson were as follows:

June 7th, fourteen days after operation. The nerve has whitened considerably; the congestion has subsided, and the tortuosity of the vessels is much less. Vision 20/50 in the left eye. The whole appearance now is of a declining retrobulbar neuritis.

June 16th. Vision in the right eye is normal, in the left

20/20. Inflammation in the left nerve was almost completely gone. The nerve is still a little paler than its fellow. Form field is normal. She matches colors readily with the left eye, but says they look less intense than with the right. Recovery appears to be almost complete.

Addendum.—The patient was seen again June 26th. The right palpebral fissure was, as before, greater than the left. The pupils were equal, large in size, and reacted well to light and accommodation. The optic neuritis had entirely disappeared, but there was a decided pallor of the disc. Vision in the affected eye, 20/20. There was occasional slight pricking sensation in the wound, but there was no abnormal sensation in the eyeball. The patient felt quite well in every respect.

This case is interesting from several standpoints: (1) because of the existence of a high degree of papillitis and visual defect dependent upon extra- and not intracranial lesions; (2) because of the indefinite and apparently mild pathological condition affecting the sinuses (hyperplasia); (3) because of the prompt and complete alleviation of all symptoms after operative interference directed to these sinuses; and (4) the rapid *restitutio ad integrum* in the functional capacity of an eye previously so profoundly diseased.

Papilledema in these cases is not always monocular, though often it is. I have seen double choked disc of five diopters resulting from a similar condition and with vision twenty-twentieths in each eye. Thus in sinus involvement we may get one or both eyes affected, while in intracranial neoplasm a unilateral choked disc with the other eye quite normal is an extreme rarity, if in fact such a condition ever exists.

The mechanism involved in the production of a papilledema in sinus disease is easily understood. The optic

nerve lies upon the body of the sphenoid separated from the mucous membrane of the sphenoidal sinus by a plate of bone varying in dimension from paper thinness to twelve or fifteen mm. thick. This relation on the one hand between nerve and sphenoidal sinus and on the other between the posterior ethmoidal and sphenoidal sinuses is often very intimate as has been shown in Onodi's excellent monograph and the papers of Loeb and Birch-Hirschfeld, in which the anatomical arrangement of these parts is worked out. Under these circumstances it is not difficult to understand how an inflammatory process affecting the sinuses may either directly by continuity or indirectly by means of the lymphatics and blood stream easily affect the optic nerve to produce the picture we observed in this case.

SYMPTOMATIC TIC DOULOUREUX

JOSEPH COLLINS, M.D.

A widow, 66 years old, entered the Neurological Institure, on September 29, 1916, complaining of intense, intolerable pain in the left side of the face which had been increasing in severity for six months. She had had some manifestations of it for a year and a half, but it was not until eight months before her admission to the hospital that it became so severe that she sought relief. The pain, which was described as "tearing," "crushing," was present continuously and at times displayed itself in excruciating paroxysms. She had been given different forms of treatment, including two injections of alcohol into the sheath of the fifth nerve, but without relief.

Physical Examination.—The patient was a stout, flabby, elderly-looking woman. Her blood vessels were sclerotic, the systolic blood pressure 210, the diastolic 160; the heart sounds and the rhythm of the heart suggested chronic myocarditis. There was no evidence of renal inadequacy measured by the phenolphthalein test. Chemical and microscopic examination of urine revealed no structural disease of the kidneys. Her digestion was profoundly disordered and she said that she was losing weight. The Wassermann test of the blood serum and cerebrospinal fluid were negative. The only disorder of the nervous system aside from hypalgesia of the left side of the face over the second branch of the fifth nerve was complete paralysis of the left external rectus. The left

palpebral fissure was much narrower than the right, the pupils responded sluggishly to light. The cutaneous sensibility supplied by the first branch of the trigeminal nerve was quite normal; the third branch of the fifth nerve was unaffected.

Dr. Culbert reported that the right nostril was completely occluded by polypi and that the left nostril was filled with pus. The right frontal and ethmoid sinuses did not transillumine well. Dr. Caldwell, who made the x-ray examination, reported that the right frontal and maxillary sinuses were entirely filled with pus. The clinoid process on the left side was very long but there was nothing found on the left side of the skull on either direct or stereoscopic examination to explain the pain. It will be recalled that the pain was confined to the left side of the face.

It was not thought that the trigeminal neuralgia was symptomatic of focal disease. We believed that profound purulent disease of the sinuses may have stood in causal relation to the degeneration of the blood vessels and probably of the Gasserian ganglion as well, that the trifacial neuralgia was an expression of the degeneration in the latter tissue. It was impossible to reconcile the findings of the rhinologist and the radiologist with the left-sided tic douloureux. Although the patient had had trouble with her nose and sinuses and although she had had headache for at least a year and a half, the symptoms of trigeminal neuralgia seemed to have existed only six months. It was realized, however, that there was no way of counteracting the disease of the blood vessels and of the ganglion without overcoming the underlying cause—the pyogenic infection. She was therefore transferred to the Manhattan Eye and Ear Hospital, so that Dr. Culbert might submit her to such an operation as he thought her

condition would tolerate. After deliberation and consultation he decided that she was not a justifiable operative risk and she returned to her home, where she gradually grew worse and died about four weeks later. Dr. James G. Dwyer, Pathologist to the Manhattan Eye and Ear Hospital, made an autopsy, and he furnished us with the following report:—

Autopsy.—The examination was limited to the skull. When the calvarium was removed, the dura was found to be thin and adherent to the bone. There were no evidences of meningitis, except in so far as the adhesions of the dura signified an old slow process probably secondary to pressure. The brain itself had quite a normal appearance, save for the profound atheroma of the blood vessels, especially at the base of the brain.

The ethmoid sinuses on the right side were quite full of inspissated pus resembling cottage cheese. The right frontal sinus was full of liquid creamy pus. The sphenoids were continuous and full of inspissated pus enclosed in a membranous sac which did not open into the nose. The left frontal sinus was clear. The left ethmoid sinuses were quite full of pus and there was an opening through the outer wall into the left orbit; and in the left orbit was found a collection of thick pus which bathed the nerves and vessels. There was no exophthalmus of either eye. There were openings from the left ethmoid directly into the left antrum and the latter was full of pus. Its posterior wall was broken down, and this allowed the pus to bathe Meckel's ganglion.

Cultures of the pus showed pure staphylococcus pyogenes aureus.

OBSESSIONS AND FIXED IDEAS

SANTE NACCARATI, M.D.

(1) A Case of Psychasthenia

Psychasthenia is the name given to a disorder of an individual which expresses itself most conspicuously by an idea or thought that possesses him and for the time being dominates him to such a degree that his activities and efficiency are profoundly curtailed. This idea or thought is commonly spoken of as an obsession. Although the obsession is the chief clinical feature of the disorder, the afflicted individual displays in addition to it, usually, a physical and mental state that may be called neurasthenia. Psychasthenia is not a very common disorder, apparently, judged by our experience in this clinic. Obsessive states of mind are probably fairly common, but when the obsession does not reach a degree of dominancy as seriously to influence the individual's conduct, to impair his happiness, or to curtail his efficiency, the possessor does not consult a physician.

One of the most typical cases that we have had in the clinic recently is herewith presented. A man 47 years of age married, when he was thirty-five, a wife much younger than he. Until five years ago his life was without particular incident, then he lost about $30,000 in business. He thinks that he did not worry about this loss, although he admits that he was very sorry for his uncle who lost about twice as much. Then he went to Florida for three

months, with the idea of settling there; but he did not like the place. Later he became a traveler,—a solicitor, until September, 1913. At that time he had a nervous breakdown; he could not sleep; he could not lie in bed; he could not rest. He thought it might be due to the strain of the continuous traveling (he had been traveling for fifteen years). A physician told him that he had neurasthenia and advised him to go to a sanitarium. He was in the sanitarium for three months, during which time he improved, but at the end of the third month he had a vision in which he saw a multitude of people in the distance, and heard a voice which said: "If this man had died, the world would be safe now. I waited for him thirty days but he did not die." It happened at about 5 A. M. Then everything disappeared; he woke up but remained in bed until seven o'clock. He did not pay any attention to it at that time. A few days later, as he felt better, he left the sanitarium and returned home in condition to attend to his business. But six months later (the vision happened in February), the war broke out, and then he remembered his vision or dream (he does not know what it was exactly), and realized that the voice he heard was that of Jesus Christ. Then he felt sorry that he had failed to fulfil his mission in this world. He felt sorry that he did not save the world, he felt guilty although he knew that he could not help it. Since then almost every week, even every two or three days, this idea came to his mind and almost dominated it—namely the idea that if he had died there would have been no war. This idea came early in the morning; it was accompanied by a state of restlessness and cramp in the stomach. It was irresistible; induced a state of anxiety in his mind; the idea would follow him imperatively, irresistibly. After having overcome it, he would feel relieved.

Physical Examination.—The physical examination showed practically nothing.

About his mental condition I might say that he is perfectly oriented; ideation, perception, memory, association, affectivity do not seem altered at all. He is aware of the foolish foundation of his idea; he does not cherish it, on the contrary he fights it. It is useless to try to convince him that his idea is false, because he knows it.

There are no hypochondrial ideas, no ideas of reference, no ideas of persecution associated with an exaggerated consciousness of the self, so characteristic in paranoid states. What the patient really shows is a fixed idea, an imperative conception brought into his consciousness by the emotional shock of a dream and developed later by the revelation of unexpected happenings.

Diagnosis.—In conclusion, the fundamental characters of the idea are the clear recognition by the patient of the unreasonable nature of it; the occurrence in attacks with varying intervals of comparative calm like a mental tic; the fight, the sensation of relief; or liberation when it passes away. I think this is a case of psychasthenia developed in an individual with constitutional nervous asthenia. Psychological study of the patient has been undertaken, methodic dream analysis being the starting point. The patient himself has asked for psychic treatment, and there is no doubt that psycho-analysis is the only therapeutic procedure which could prove successful in this case.

(2) Fixed Idea—Hysteria or Dementia Precox

An American girl, 18 years of age, had been well and happy until shortly before she came to the hospital. She came, she said, to be relieved of a delusion or obsession of

a peculiar smell around her. This smell follows her wherever she goes, but it is especially bad when she is in a closed place, or when she finds herself in a crowd. The only time, she says, this smell does not trouble her is when she is at home with her people. It makes her very unhappy, for she is convinced that the odor emanates from her body. She knows it does not; nevertheless it seems to come from the body. She is a clean girl, she changes her underwear twice a week, and she does not perspire much. None of her friends ever made an allusion to this smell, so she thinks it is absurd and ridiculous that she should have this fixed idea in her mind.

As a result she has tried to avoid public gatherings and her friends, and has almost developed a fear of crowds and closed places. However, this is not a real claustrophobia.

Lately she has been using perfumes, but to no purpose. The smell has become even more oppressive.

She was a salesgirl in a department store, earning nine dollars a week, and although she did not find her daily work harder than usual, she noticed a certain aversion to going to her working place, or among her companions. The smell, which constantly persecuted her, troubled her so much that two weeks ago, according to her relatives, she became hysterical, and had to leave her position. It seems that by the word "hysterical" was meant a typical hysteric fit, which occurred at that time and which was followed by another after ten days.

She has tried all means to get rid of this trouble, but in vain; and she is convinced that, once rid of it, she will be cured.

Physical Examination.—The physical examination did not show anything of importance; she was a healthy looking girl, well developed and well built. No cases of nerv-

ous and mental diseases existed in her family. She showed distinct Rosenbach's phenomenon.

When I tried psycho-analysis on her she gave the following account of the occasion on which she first was troubled by the odor: "About two or three months ago, I was going home in a closed and crowded street car, and at that time I noticed a very disagreeable smell of fried lard which disgusted me. It emanated from somewhere in the car and continued until I left it. I went home, however, and was not troubled until a month later. Then I was walking on Broadway and 40th Street, and suddenly I smelled something disagreeable around me, which has never left me since. I went in the subway and had the impression that the smell emanated from my body, but I perceived that none of the crowd showed any signs of smelling it."

I asked the patient whether she had the sensation of that smell in my presence and she denied it, admitting that she did not smell it when her mind was distracted. She was unable to give the nature and character of the smell, which she said was indistinct and yet never passed away.

Her mental state, apart from the hallucination of smell, was quite normal. She was perfectly oriented and she did not show any disturbances of ideation and memory. Affectivity and association were not altered at all. The fact that she did not like to go with friends and that she avoided public gatherings must not be interpreted as an act of apathy or indifference, in other words, as a disturbance of the effective life so characteristic of schizophrenic states. It was rather a psychic defense reaction, or, as Freud calls it, a mental protective mechanism, a means of fighting her perceptive disorder by avoiding the circumstances which, in her experiences, induced it

(crowd, closed place, etc.); or as a fear that others could discover it.

Her dream analysis showed that during the last two months she dreamed several times that she was running in the streets alone and that she was swimming and breathing fresh air.

Diagnosis.—In analyzing this case, I would say that the diagnosis of hysteria and that of psychasthenia should be taken into consideration. I am inclined to exclude the latter for the hallucinatory character of the idea rather than for the obsessive, for the almost constant persistency of it rather than its occurrence in attacks with intervals of quietness, for the absence of the fight with the consecutive sense of relief or liberation; and finally for the suggestibility of the patient. We may exclude the schizophrenic group, since (1) there is no loosening of the associations and no sudden arrest of associative actions; (2) there is no change in the feelings to apathy and indifference; (3) there is no tendency to ambivalence; (4) there are no alterations of the more complex functions, as Bleuler states, viz.:

(a) Autismus is not manifested.
(b) Attention
(c) Will } show no changes.
(d) The Person (the ego) does not show a tendency to cleavage.
(e) Intelligence
(f) Behavior } seem normal.

Taking up the theory of the mechanism of hysteria of Freud, we can explain this case easily.

Freud, admitting that in normal individuals, certain temporary dissociations, which he calls psychopathology of everyday life, take place, found that the same mechanism underlies the complex pathological state of hys-

teria. In normal everyday life, disagreeable or painful thoughts are always forgotten; we intentionally, or even unconsciously, push them out of consciousness, so as to free ourselves from disagreeable feelings or pain. This may be called a mental protective mechanism. Sometimes, however, a disagreeable incident remains in our unconscious memory, forming what Freud calls a complex. Then, because we have no control over it, this complex acts in a pathological manner. It cannot run its normal course, and, therefore, it becomes converted or changed into a condition which we designate as hysteria.

I think Freud is right when he opposes Janet, who says that "any sudden emotion may cause hysteria," and affirms that "only those emotions or ideas cause hysteria which are painful and which the subject has difficulty in expelling." In our case the fact that the patient was obliged to stay for some time in a disagreeable atmosphere—a closed and crowded place from which emanated a bad smell—represents the painful emotion which remained in her subconscious memory and which could not be expelled.

In her dreams we see also the fight for expelling the painful experience. Be the dream, as Freud says, "a symbolic veil for repressed desires," or, as Jung says, "the subliminal picture of the psychological condition of the individual in his waking state," we clearly see in her running in the fresh air and in her swimming in the fresh, clean water the representation of her mental state —her desire to be alone, not in a crowd of people, in which surroundings she first experienced the bad odor, nor in a closed place where odors are formed. The running and the swimming symbolize the movement of the car in

which she was running at the moment that the emotional shock occurred.

Freud reported a similar case which he characterized as hysteria. It related to a governess, who was troubled by the persistent hallucination of the smell of burnt pudding. Psycho-analysis showed that once while she was playing at cooking with the children of whom she had care, a strong odor spread through the room. The children had left the pudding which they were cooking, and it had burnt. Ever since, the smell had pursued her. Further examination revealed that at the bottom of the psychical excitement was a sexual repression.

Whether in our case the painful experience had a sexual coloring has not been investigated. It is likely that at the time of the original emotion, the patient was in a state of abstraction or, as Freud calls it, in a hypnoidal condition during which the impressive incident became immediately dissociated from consciousness, but the symbolic emotion was preserved in the subconscious mental life.

Of course, in a condition like this, if the dissociated experience be synthetized with consciousness and thus brought under control and censorship, a decided therapeutic effect should follow.

The treatment of this girl, therefore, will be distinctly psychotherapeutic, without neglecting, however, the physical element of treatment through baths, electricity and rest.

(3) The Psychosis of Imperative Ideas

Phobias, obsessions, impulses were for a long time included among the "monomanias" of Esquirol, and later classified by Morel under the general name of "emotive delirium."

It was Westphal who first called them compulsory representations, after Falret had made his valuable contribution to the subject under the caption of "Folie de Doute."

Arndt, E. Morselli and Krafft-Ebing considered the fixed ideas in relation to intellectual disturbance and gave them the name of "rudimentary paranoia," as they are an expression of a constitutional morbidity and usually assume a paranoiac character like a systematized abortive delirium.

In many modern text-books the psychosis with fixed ideas is not described because the authors, following Janet's conception, have classified it under the name psychasthenia. Janet's teachings have not, however, been generally accepted. The Italians, for instance, maintain that the psychosis with imperative ideas is not connected with simple degenerative psychoses (constitutional nervous asthenia), but bears a closer relationship to the dementia precox and the manic-depressive groups.

One of the most typical pictures of this clinical form is the following:

A girl, eighteen years of age, came to the hospital complaining of "evil imaginations," which dominate her mind against her will. These imaginations, which she is either unable or unwilling to explain, and which are in conflict with the ideals of her personality, very often come to her mind and possess it to such an extent as to curtail her activities and interfere with her happiness.

She tries to fight them in every way, by distracting her mind, by going to church, etc., but it is useless; they persecute her just the same.

Her mother relates that she has not noticed any change in the character of her daughter, as she always was a good and quiet girl, rather religious, not fond of friends

and amusements. She prefers to go to moving pictures and to read papers and romances. She has no father; her foster father is a drunkard and sometimes brutal to her.

Physical Examination.—Physical examination does not show anything of importance. Her appearance, her nutrition, her circulation seem good; there has been no mental disease in her family.

Mental Examination.—The mental examination shows that association, memory, affectivity are normal and that only ideation is disturbed.

The ideas, of which she seems possessed, have the following characteristics:

First—Patient has the consciousness of her morbid state, but, although she fights, she cannot get rid of the ideas.

Second—They are irresistible; patient is unable to see their frivolous foundations.

Third—They are associated with the emotional disturbance of anxiety, which is determined by the conviction of the impossibility of overcoming the same.

Fourth—They induce in the patient's consciousness a fight for the purpose of expelling them.

Fifth—They are followed by a sensation of relief, when they are momentarily discharged.

Further investigations have shown that these ideas developed soon after a dream, which acted upon her mind as a real emotional trauma.

"It seemed," she relates, "that I was in an unknown place, where I saw a man with a black mask, a man who wanted to hypnotize me, but who did not succeed. I left that place, but the man followed me all over, trying to catch me. I found myself in a cabaret, where the same man appeared. I fought to escape from him, and some girls, who were there dancing, asked me to go with them

to a fortune-teller. I told them that I had no faith in fortune-tellers, but nevertheless I went with them. When I was in the street, however, the girls disappeared, and I found myself in front of a church with a young man whom I did not know."

I think in this case the original Freudian theory may be applied very well. In this girl a sexual emotion which is in conflict with her tendencies, with her education (subject as she is to the moral and social restrictions), has been confined in her subconsciousness and transformed into a real "complex."

In analyzing this dream, we clearly see in the man with the black mask, the man who tries to hypnotize her, the sexual libido, which has been following her with all kinds of temptations.

The common dancing place, the cabaret, where she found herself afterwards, the fortune-teller's place, where other girls wanted to take her, have a reputation, whose significance in this case needs no explanations.

The church, where she found herself with the young man, is the right place for a good girl, a place through which a girl of morality should pass in order to satisfy her sexual desires.

A CASE OF DEMENTIA PRECOX, SIMPLE DEMENTED FORM, OR SCHIZOPHRENIA SIMPLEX

HENRY W. MILLER, M.D.

At a recent conference, Dr. Collins, in presenting a case of dementia precox, laid particular emphasis upon the fact that errors in diagnosis of dementia precox are relatively high. I think there is no question but that the statement is correct both in the making of the diagnosis when the disease does not exist and in failing to recognize the disease, particularly in those cases in which there is an acute psychosis which passes off, leaving the patient practically in normal mental health. We are rather prone to question our diagnosis of dementia precox when the patient does not show some sign of deterioration, in keeping with the name of the disease. In spite of the many errors of diagnosis, we are nevertheless dealing in dementia precox with a very important group of mental disturbances, and we must attempt to define clearly the great variations in the clinical pictures. This case is presented to-day to show a typical dementia precox process, not because there is anything unusual in the clinical picture.

The young man, who is 26 years of age, has a rather interesting family history. His forbears beyond his parents were evidently of fairly good stock. His mother was of a neurotic make-up, while his father was an alcoholic with a very irritable temper, possibly alcoholic in origin.

The patient was the sixth child in a family of nine, one brother of which was alcoholic and was said to have died in a state of extreme mental excitement. Another brother was a persistent alcoholic and two other brothers and a sister were decidedly neurotic. Thus we see in this family a most decided constitutional weakness. The patient in early life was evidently normal and made normal progress in his school life. At the age of twelve he had diphtheria, followed by an attack of inflammatory rheumatism and later by what was unquestionably Sydenham's chorea, which continued for about two months. During his later adolescent life he began to manifest the symptoms which we have come to look upon as rather characteristic of an oncoming dementia precox. He became rather shut in, found his studies in school increasingly difficult and did not advance as well as he had done in earlier life. He also became addicted to alcohol, very probably an indication of the beginning of this deterioration process. He became somewhat of a day dreamer; he gave up his studies before he had completed his high school course and took up the work of a draughtsman. In this he was never very successful, nor did he ever hold one position more than a year at a time. There have been no emotional complexes discovered. He drifted along, becoming rather more seclusive, more given to his day dreaming until about two years ago, November 4, 1914, when he became unable to concentrate his attention on his work and is said to have been somewhat confused. There was never, however, any very distinctive delusional trend. In March, 1916, he was committed to the St. Lawrence State Hospital, and it is quite evident from their records that his condition there was practically identical with what we see at the present time. They report that he reacted well to the hospital routine, talked freely

and spontaneously, showed no restlessness, was neat and appreciative; denied auditory hallucinations, was well oriented, had a good grasp of occurrences in the surroundings, retained school knowledge well and had no delusions.

They report that he took little interest in anything and read very little. He had episodes of restlessness when he was most abusive in his language, but he soon became quiet and on the solicitation of his family was allowed to go on parole on the 1st of July. He was admitted to the Neurological Institute on October 31, 1916.

Physical Examination.—Physical examination shows nothing significant except a lack of hair upon the face and over the body generally. His symptoms are very much as described above. The most marked symptom is without doubt the emotional indifference. In spite of the fact that he has a certain amount of insight, recognizing that his mind does not work right, he is quite indifferent as to the present situation and as to the future. The next important symptom which he presents is a certain dilapidation of thought, which is best shown by some of his writing.

With his emotional weakening he has a decided dulling of the initiative and a judgment defect. This latter symptom, while it may not be well demonstrated by ordinary tests, is best shown by his practical experiences in life.

The significant feature is the presence of the characteristic deterioration, without any delusional trend, but with the retention of memory and school knowledge.

The case is presented as a classical one in a series of dementia precox cases for the purpose of outlining clearly the symptomatology in one group of this type of psychosis.

Diagnosis.—The clinical picture according to Bleuler would be classified as a schizophrenia simplex which he has clearly defined in Aschaffenburg's "Handbuch der Psychiatrie." His analysis of the group of schizophreniæ forms an important contribution to modern psychiatry with which we should be familiar. The name "schizophrenia"—"a splitting up of the mind"—significantly characterizes the disorder. Bleuler, in describing the simple form of schizophrenia, uses the words of Clouston: "The patients become affectively and intellectually weak; their will power becomes lost, their ability to work becomes diminished, they appear dull and finally present the picture of a pronounced dementia."

Bleuler's analysis of the fundamental disturbances of the associations and the affects is especially emphasized in his contribution. He considers the schizophrenic group to be most clearly characterized by disorders in the field of the associations and the affects, and from his analyses we can obtain many helpful suggestions.

AN UNUSUAL CASE OF DEMENTIA PRECOX

SANTE NACCARATI, M.D.

The patient, a Russian Jewess, 24 years old, came to this country when twelve years old.

She was married when 21 and had two children and no miscarriages. According to her statement and to that of her brother there are no cases of nervous or mental disease in her family.

Her brother says that she lost about 40 pounds during the last six or seven months, but she does not look emaciated. This loss in weight together with insomnia, fatigue, and slight gastro-intestinal disturbances, were the chief symptoms. About five months ago, according to her brother, her conduct became strange and unusual. She developed the belief that neighbors were trying to alienate her husband's affections from her. She said that they were conspiring against her with her husband. They accused her of being a bad woman, of not taking care of the house, of not being clean, etc. She did not pretend to understand the persecution to which she was subject, and she became very suspicious of people and things. Some of the episodes which were related of her are:—

(1) About four or five months ago, she went to see one of her brothers, to borrow some money from him. After she returned home she had several crying spells and became very angry, saying that he was her enemy because he had refused to give her money; in reality he had given it to her.

(2) Another day she went to her husband's store (a butcher shop), and seeing a woman there she drove her out. The explanation which she gave was that the woman had told her husband that she (the patient) was dirty, was not a good woman, and did not take care of him and of the children. Another day she chased another woman out of the store, because she heard her speak to her husband against her.

(3) Once, she says, her husband and her mother-in-law, believing the accusations of her enemies, told her that if she did not take care of the house and be a clean woman they would send her away.

(4) She thinks that there are four women neighbors who are her most stubborn enemies, and who accuse her of moral shortcomings to her husband to such purpose that she has lost her husband's affection.

It is not an idea of jealousy which she has developed, but rather ideas of reference and of persecution. This delusion of being persecuted, however, is not associated with pathological egocentrism. As a result she has become very suspicious; she goes to her husband's desk and looks at his letters, his papers, his bills; once her brother saw her pick up an envelope, and after looking at it several times, say: "Somebody wrote a letter to him."

Little by little she has become very slow in dressing herself. Sometimes it takes half an hour or more. When she goes marketing for her little household, she goes several times, bringing one article each time, while she could have brought them all at once; meanwhile her memory seems quite good.

She gets tired easily. She does not take interest in her house as before, and she does not like to associate with friends or neighbors.

Sometimes she starts a phrase, without any meaning, and stops suddenly in the middle of it, without completing it, and passes to the expression of another idea. For instance, once she suddenly said to her brother, "There is a big war in 136th Street" and then did not go on, and would not explain it. This symptom of loosening of the associations and sudden arrest of associative actions so characteristic of dementia precox is very prominent in this case.

There are no mannerisms, no stereotypies, no hallucinations, and no primary disturbances of perception, orientation or memory are demonstrable.

Diagnosis.—The lack of interest and initiative, the bizarre actions and conduct induced by her ideas of persecution and references, the disturbances of association and affectivity (will), the limitation of her contact with the external world (called "autismus" by Bleuler, "loss of the sense of reality" by Janet), without any disturbance of perception, orientation and memory in a young subject, together with the physical signs of loss of weight, insomnia, fatigue, occurring without attributable cause, in a young woman who has never displayed evidence of mental disorder or of adult infantilism, are sufficient symptoms for making the diagnosis of dementia precox.

THE PARANOID FORM OF DEMENTIA PRECOX

JOSEPH COLLINS, M.D.

The paranoid form of dementia precox is of much less frequent occurrence than either the simple hebephrenic variety or the catatonic. A partial explanation of this may lie in the fact that when the paranoid form of dementia precox is not associated with mental enfeeblement, that is, when there is complete retention of consciousness, many of these cases are often classified as paranoiac. The case about to be related is so classical, and illustrates the characteristic features of the disease so admirably, that no other attempt will be made to convey the salient features of this variety of dementia precox than the citation of the case.

The patient is single, an Irish-American, forty-one years of age, and an employee in the Foreign Department of the Post Office. Until the beginning of the present illness, he had been singularly well. As a boy in school he was considered very bright, affable and friendly. He became a choir boy in the Episcopal Church before puberty, and for twenty years was a member of the choir of one or another prominent churches in New York. He always prided himself on his morality, and he denied with scorn that he had ever infringed upon ordinarily accepted sex conventions. In fact, he added voluntarily to his stature when this subject was being discussed with him; that is, he drew himself up to a very considerable height and delivered himself most impressively of his convictions

concerning onanism and illicit sex relations. According to his mother's estimate he was one of the best boys that ever lived, and he had no hesitation whatsoever in agreeing with his mother. In fact, he prided himself upon his appearance, upon his physical and mental possessions, and upon his conduct.

The history of his illness seems to have been about as follows:

On August 7th, 1915, he decided to take a holiday. He went to Asbury Park with some friends in a cheery, happy frame of mind. He had scarcely arrived there when he became conscious of the fact that he was being shadowed, and he determined at once to go home. When he got into the train he noticed that men dressed in blue went down the aisle, some of them putting their hands around towards their "pistol pockets" in a suggestive manner. He did not know the meaning of this and felt scared and apprehensive. On the steamboat he noticed other men acting in a similarly suspicious way, and he sought an interview with the captain of the boat to apprise him of his fears. The captain reassured him and this assurance was accepted in a measure, but in reality it did not relieve his mind in any way. When he reached home he was in a condition of extreme fear and apprehension.

The next morning some one rapped on the door of his room and he heard a voice saying "Do you want anything from us?" to which the patient replied, "No, certainly not." Then the intruders went away. He believed that they were undoubtedly thieves, or the members of some thieving society. They did not annoy him any more that day.

After remaining home a few days he resumed his work in the Post Office and was at work until October 17th of

the same year, during which period he was not bothered at all, nor does he recall now (August 2nd, 1917) that he was frightened or apprehensive at that time. It was about October 17th, 1915, that he became very ill and was again frightened and influenced by forces which he believed probably represented the agency of the Catholic Church.

He was reared carefully in the Protestant faith, was a God-fearing man and had no desire to encroach upon the beliefs of others, or to tamper with their religion, and it was therefore a matter of profound astonishment to him that the machinations of the Catholic Church should reach out and get him. They began to influence him in various ways—to talk about his mother and make very definite accusations against her, reflecting upon her conduct, but not upon his relationship toward her. They were bent upon convincing him that the house in which his mother was living was of extremely bad repute. He told this to his mother and she tried to convince him that the house harbored only average, honest, intelligent people, but he knew better and was sorry that he could not convince his mother that it was the Irish who knew that he and she had some money, and were after it. He soon realized that it was a neighbor, Mrs. ———, the bookkeeper at the ——— Club, who controlled his mother's mind. This Mrs. ——— had stolen fifty dollars from the house, that is, as far as he knew, and it was his honest belief that she did, and he had reported it to the police station where he was told that nothing could be done in the way of recovering it. It was a very extraordinary thing that as soon as Mrs. ——— got his money she dressed in blue, which of course meant that she had gone into the government service. There were two kinds of persons in that service; a woman's underhand service—a sort of secret alliance—and the man service. This Mrs.

——— had another woman, a Mrs. ——— come down from Rome, New York, and between the two of them they rearranged his household. They put things right and left; for instance, they would arrange things studiously on the dresser in certain groupings, the significance of which he was unable to fathom.

He could not tell his mother everything that transpired and could not make her understand how bad the house was, and how treacherous were their neighbors, and this preyed upon his mind until he finally became very ill. He went to the St. Luke's Hospital and asked for a room, but they would not admit him and referred him to the Vanderbilt Clinic, where electricity was applied to his head, through a sort of cap, and since that time he has not been worth a nickel. He realized now that this was a part of the work, an initiation of the procedure to which he has been subjected.

He moved downtown, and there they began their work upon him systematically and earnestly. As he lay in bed the people who occupied rooms in houses around him and opposite, owned by the Catholic Church, sent wireless messages across, which he received through his forehead. Oftentimes they were suggestions; at other times they were articulate statements urging him to tell what was being done in the Post Office. They suggested that he put himself out of the way; they reproached and taunted him because of his incest; they told him about his mother's infamy. At times they turned him over in bed; he habitually slept on his left side, and one time they turned him abruptly from his left to his right side. That was the way they had of changing a man's religion. He realized that there was a cabal in existence to kill him and to get his money. He had made an arrangement with his mother that if he died first what little money

he had was to go to her, and if she died first her money would go to him; so in all probability this cabal had connived to get them both. It was perfectly clear to him, as it must be to any one who was not blind, that they were boring into his mother's eyes now with electrical instruments, the object of this being to accomplish her destruction. Meanwhile they hoped, perhaps by suggestion, to make him go off and kill himself. In this way they would accomplish their ends.

The machinations of these people were by no means limited to their operations upon him and his mother, but they extended throughout the country, and even the government did not appreciate the extent of it yet. He did not know just how to go about communicating it to the government, so that the weight and importance of the struggle which was going on would be adequately sensed. In reality, he believed that at the present time they (the cabal) had got hold of the United States government and were sending all these men abroad, ostensibly as soldiers and to enter the war, but in reality to get rid of them. He could not explain why the cabal wanted to get rid of them—just to get rid of them, that was all—so that it would be easier to control the country.

Aside from having gone to the police and having asked for protection, and having written to the Department of Justice, the persecutions had not yet influenced his conduct. Several days before he was first seen by the examiner, the patient had telephoned to police headquarters and two men had been sent up to the janitor to see what was the matter; but the police did not come to see the patient although they knew who had telephoned, for the patient had given his name and address.

He was not afraid to go on the street alone; he was

still going to church, but did not sing any more; his voice had given out about June first.

His story was told with much earnestness and with infinite detail. Oftentimes he insisted upon a minor correction being made, stating that it was necessary to get matters perfectly correct. He drew himself up to his full height, inflated his chest, gave his delivery with proper intonation, and presented the whole matter with an air of satisfaction, of determination, and of earnestness. A certain dramatic atmosphere was created by him as he related the foregoing narrative. Such remarks as "Now, mother, don't butt into this—this is a man's work—men are now in conference about a serious matter" were frequent. He smacked his lips, looked earnestly at his interlocutor, and had the satisfied expression of one who, having formulated the outline of a great work, saw the first part of it satisfactorily begun.

He believed there was nothing to conceal in his story, nothing of which to be ashamed, nothing that he did not wish to have investigated to the fullest extent. Indeed, he felt that this was the first time he ever had had an opportunity of earnestly enlisting the sympathies and interest of any one who, according to his statement, was worth while.

His mother remarked that he had become fretful and pre-occupied, but he believed his mother did not understand this matter, and he had really almost despaired of ever being able to get her to grasp the enormity or the extent of it. All that he wanted was protection, so that his mother and himself might live where they were in safety.

To his mother's statements that he had become forgetful, reticent and silent, he replied that he had much to think about.

When a light was flashed into his eyes to test the re-

sponse of the pupils, he exhibited great concern and said this had been done at St. Luke's Hospital and had had a very bad effect upon him. He then expressed a desire to go home at once, stating that he would return the next day. Meanwhile he examined his eyes in a mirror with much solicitude. As he left the room he turned for a moment to say, "Well, doctor, I expect that you will get this matter on to Washington now, right away," and his face assumed the expression of a bishop pronouncing a benediction.

The following morning the patient returned, together with his mother, and the urgent necessity of a rest and change was explained to him. To this end he requested that a letter be forwarded to the Post Office authorities, as he was still regularly employed; this was done and he promised to report as soon as matters could be arranged.

On August 6th, 1917, four days after the first interview, he reported again, at this time for admission to the hospital for a few days' investigation of the persecution from which he suffered. He related that since his last visit the persecution had been almost constant; for instance, he was unable to remain in his house at all the previous night. He went to a hotel to sleep and while there was free from any persecution. He was fairly certain now that those responsible for his suffering were some persons in the rear of the house he occupied; they were no longer in the front. The voices that he heard were female and whispered continuously, "Your mother is sick—your mother is sick—your mother is sick." If he were well he felt that he would get on a train and go somewhere—any place in the world—and rest in the air, and so secure freedom from this persecution. He felt that it would be better if he took such a trip with a friend —some man—so that he might have company because, as

he related, he did not hear any of these voices when he spoke; it was only when quiet that he was so persecuted. Despite this fact the voices were very real.

When asked if he slept much the previous night, he replied "from three o'clock." When told that his mother remarked that she walked the street after him, and when asked where his mother went, he stated that he did not know and replied—"She is afraid and don't understand anything about this—this is a medical case."

Upon further careful questioning it was ascertained that he had been in the psychopathic ward of Bellevue Hospital in 1915 for three days, but that previous to this time he had never "heard" things; this he readily explained by the fact that he worked in the Post Office and was kept busy all the time. It was in 108th Street (an earlier residence) that he suddenly got one of these messages to put his mother in a sanitarium. "The walls were wired or something." However, he paid no heed to these messages.

His mother related that on the previous day (August 5th, 1917) she interviewed two policemen who told her that her boy had been talking to them about this matter for the past two or three months, but the patient had mentioned it to so many policemen that he had no definite knowledge of the officers referred to by his mother.

A simple brief narrative of the causes and sequential incidents leading up to the present war was given and the patient requested to repeat this or state the substance of the narrative. To this he replied that he did not know anything about those things, offering the explanation that the continuous severe strain under which he had lived had prevented him from comprehending details.

The patient was admitted to the hospital and promised faithfully that he would remain and conform to all the

regulations. He wanted to enter, and felt that he would be better off here than anywhere else. Nevertheless, within an hour of the time of his admission, he walked out of the hospital, despite attempts to persuade him to remain.

Subsequent History.—Two days later his mother called and said that he went from here to a hotel where he remained overnight, and returned to his home the following morning in a highly excited state. The mother was very much frightened, as he acted "wild-like" and gave indications of great violence.

A few days later he was legally adjudged a lunatic and the report from the superintendent of the hospital in which he is confined sets forth that he is rapidly dementing.

GLIOSIS OF THE SPINAL CORD

C. BURNS CRAIG, M.D.

If the incidence in this country of gliosis of the spinal cord has not increased, the readiness with which it is recognized certainly has. Practically unknown in American medical literature twenty years ago, the symptomatology of syringomyelia is now fairly familiar to the average practitioner. Despite this general familiarity with spinal gliosis, I am convinced that its occurrence is more frequent than is commonly believed. It is not uncommonly confused with disseminated sclerosis, chronic progressive muscular atrophy or some other disease of the motor tracts.

The following cases appeared in the Clinic of the First Division of the Neurological Institute in the same week. They represent characteristically, the symptomatology of gliosis at either pole of the spinal cord. Both patients were strong, vigorous men in the second decade of life.

Case I—Of the Medulla and Cervical Enlargement

The elder patient, 27 years of age, a life insurance agent, complained of dizziness, difficulty in swallowing, difficulty in enunciation, a feeling of coldness in the left side of the face, double vision in looking to the right and disability in the use of the right arm and leg. His previous history records a fall, when 7 years old, in which he struck upon his head. He was said to have been un-

conscious for several minutes and dizzy afterwards. He passed uneventfully through measles and diphtheria. Following scarlet fever he suffered from swelling of the legs for some time. He denied venereal infection.

The present illness began about 6 years ago. While throwing a baseball with all his force he suddenly felt a sensation in the back of the neck as though some one had pulled out a handful of hair; although he stopped playing he felt no further discomfort. Three days later he had a "fainting spell." On the evening of the same day, half an hour after eating a hearty supper, he was seized with hiccough which lasted three days, during which time he remained in bed. When he got up he noticed that the right side of the body was weak and that the mouth was slightly drawn. There was no headache, dizziness or difficulty of speech. The facial weakness disappeared in four hours. In about 6 weeks the hand and arm were quite all right, save for a certain numbness and clumsiness of the right hand which never disappeared. He walked at this time as well as he ever did. Soon after this he began to grow stout, but began to train, and his weight receded from 210 to 135 pounds in two years. About this time, i.e., three years ago, he noticed that whenever he got nervous, tired or embarrassed the right leg would tremble. Soon after, this extremity was observed to be stiff at times, especially when arising in the morning or after sitting for a prolonged time. The trembling gradually increased until he could no longer control it.

Following his father's death in October, 1916, the patient became quite nervous and subject to general tremor. On December 27th, 1916, he came from work very much fatigued and found he could swallow only a small quantity of fluid and even this required considerable effort. He was confined to bed following this for a period of

three weeks. His illness was accompanied by a rising temperature and was pronounced "grippe" by the family physician, although there was no pain, sore throat, cough or nasal discharge. At the end of three weeks on attempting to arise he found the right arm and leg were partially paralyzed. He had some difficulty in articulating, but there was no aphasia. After that time he gradually improved. There was no history of nausea, vomiting or headache.

Physical Examination.—Physically the patient was rather an obese man, with slightly under-developed genitalia. In standing he spread the feet and was slightly unsteady. In walking, the right leg was noticeably stiff, and it had an outward swing, the toe scraping the floor. The right arm was held in a stiff, semiflexed position with the fingers curled in. Examination of the cranial nerves showed a hypesthesia to pin prick over the distribution of the fifth pair of cranial nerves, much more pronounced on the right side, but unquestionably present also on the left. The left side of the face was anesthetic to hot and cold and the right side was hypesthetic. There was slight weakness of the right side of the palate; the tongue deviated to the right and the mucous membrane lay upon it in folds.

Examination of the motor system showed a spastic state of the right arm and leg. The tendon reflexes of these extremities were exaggerated. The Babinski sign, the Hoffman sign and a patellar and ankle clonus were obtained on the right side. The right abdominal reflex was diminished and the epigastric absent. There was no demonstrable atrophy. The sensory examination of the body showed an anesthesia to cotton wool and to pin prick over the right lower jaw, the right side of the neck, the front and back of the right shoulder, and the radial

portion of the right hand; and hypesthesia over the radial surface of the arm and forearm. The response to hot and cold showed anesthesia over the entire neck and chest, front and back, above the axillæ, over both shoulders and the radial border of the arm, forearm and the ulnar border of the hand. There was hypesthesia over the left side of the face and radial border of the right hand.

The pupils were equal, reacted promptly to light and accommodation and there was no nystagmus. The speech was slow, rarely dysarthric. There was no definite visceral disorder. The systolic blood pressure was 130. The spinal fluid and the serum were normal.

Diagnosis.—In brief, the patient presented a right hemiplegia including disorder of the 5th, 7th, 9th and 12th cranial nerves and dissociation of sensibility over the entire face, the upper thorax and the right arm and hand. The lesion to cause this display of symptoms must be centrally located and extend from the pons through the oblongata and cervical segments of the spinal cord as far as the first thoracic segment. It must be in the center of the cerebrospinal axis. Its probable basis is a gliosis and the case comes under the caption of syringomyelia.

The only other disease which could produce the symptom-complex similar to this is disseminated sclerosis, and the question is, which of these two processes constitutes the anatomical basis of the disease. The incidence of the disease and its course are rather characteristic of disseminated sclerosis. In that disease painful paresthesia is frequently one of the very early symptoms. Syncope and hiccough are rarely symptoms of the disease either in the beginning or during its course.

The crux of the decision really hinges on the sensory disorder. While it is admittedly possible that a sclerotic process could cause such sensory alterations it is far

commoner for them to be the result of a proliferating activity of the neuroglia, in other words, a gliomatosis.

Case II.—Of the Lumbar Enlargement

The second case, a young man 24 years of age, a machinist's helper, complained of difficulty in the use of the left leg, difficulty in holding the urine and impotency. He denied venereal infection and illness in childhood. He had had tuberculous glands removed from the neck in 1912. The present disease began in December, 1914, and had been progressive. The first symptom was numbness in the right lower extremity, chiefly noticeable about the hip, extending down the outer thigh and leg to the toes. Then, gradually, weakness developed in the left lower extremities. This lameness gradually increased so that the left foot began to drag in walking and the patient would trip and stumble and occasionally fall. The numbness in the right lower extremity greatly increased, so that he could strike it, as he said, with a hammer, and not feel it. After a time this ameliorated. Imperative urination with occasional incontinence of the bowels developed and potency failed. His disability became so pronounced in about twelve months that it became necessary for him to use a cane in walking.

Physical Examination.—Physical examination showed a tendency in standing to lean to the left, but he stood securely. In walking he swung the left leg, the knee stiff, in a semicircle, the toes sometimes scraping the floor. There was also slight spasticity of the right leg.

There was general atrophy of the left lower extremity from the gluteal region down. There was evidence of weakness of both legs, particularly the left. The tendon jerks at the knees and ankles were exalted and there was bilateral Babinski sign and clonus of the patella and

ankle. The tendon jerks of the elbow were present and normal. There was no visceral disorder. The blood serum and spinal fluid were negative.

The cranial nerves exhibited no disorder. The pupils were equal, responded promptly to light and in accommodation. There was no nystagmus.

Sensory examination showed hypesthesia to cold and heat and to pin prick over the outer surface of the right thigh and hip and the entire leg and foot. The latter sensory change amounted almost to anesthesia over the right leg.

Diagnosis.—This case presented, then, moderate spasticity of the lower extremities, partial incompetency of the sphincters, weakness of the left lower extremity and dissociation of sensibility in the right lower extremity,—the Brown-Séquard syndrome plus spasticity.

On this evidence one is justified in saying that the lesion in the lumbar enlargement is bilateral and involves the motor tracts of both sides, the anterior horns of the left side and the sensory tracts on the right.

The symptoms of this case are less diffuse than those of the preceding case, but indicate a lesion similarly disastrous to the region of the cord involved, and resulting in the same symptomatic display, namely, weakness, spasticity and sensory dissociation.

The course of this case is more typical of syringomyelia. As there is no pathological sign above the waist, a diagnosis of disseminated sclerosis cannot be successfully defended. The localized character of the lesion is, to be sure, suggestive of a spinal cord tumor. The absence of pain and spinal tenderness militate against the diagnosis of an extramedullary tumor. If the concept be that of an intramedullary tumor, it is most probably a glioma, which is our original hypothesis.

HYDROMYELIA

EDWIN G. ZABRISKIE, M.D.

The formation of large cavities in the spinal cord and the manner in which these interesting lesions may develop and remain unsuspected for years, only to appear later under the guise of other pathologic states, has for many years stimulated the interest of clinician and pathologist alike. As in many other distortion phenomena the pathogenetic factors vary greatly and many occur either within or without the cord proper. Simple dilatation of the central canal into a hydromyelic cavity is found most frequently associated with hydrocephalus and spina bifida. In these cases it usually results from single increase of pressure of the cerebrospinal fluid, in a manner similar to the experiments on pressure changes in the fluid, and injections of blood into the cord by Rosenbach, Eichhorst and Lepine. Extramedullary pressure on the central canal may also produce dilatation. Lehermith and Bovière demonstrated this in a case of exostosis of the basilary process of the occipital bone in which compression of the central canal produced a cavity extending to the tenth thoracic segment. They likewise occur occasionally in cases of spondylitis. Local pressure from contracting bands of connective tissue is capable of creating a cavity and a small focus of edema. Small cavities of this nature are rather frequently seen in pachymeningitis hypertrophica cervicalis, and occasionally they develop into large cavities extending over several

segments. They usually begin as small focal points of edema from obstructed circulation and push out along the lines of least resistance in the posterior horns. One can readily imagine how this enlargement depends upon the amount of pressure and the resistance of the tissues.

Within the cord, many conditions may arise to bring about cavity formation. It may result from acute toxic myelitis, ependymitis of the central canal or degenerative changes in the blood vessels. Indeed, many are of the opinion that ependymitis and syringomyelia are always the result of the latter and that the changes in the nervous tissue are secondary in character.

One of the most interesting features of the cases of cavity formation is the manner in which they conceal their identity or mask it under the cloak of an entirely different clinical picture. Even now they infrequently remain unsuspected throughout life and are only revealed at the autopsy. On the other hand, the true extent and location of the lesion may often be completely obscured by the paucity of symptoms and signs. This is well illustrated in a case of enormous dilatation of the ventricles, ita and central canal of the cord, published by Alfred Gordon. The clinical picture indicated profound cerebral and spinal involvement, but gave no hint of the extensive lesions found in the bulb.

In rare instances the development is so insidious and hidden by vague symptoms referable to the viscera rather than the nervous system that considerable time may elapse before the involvement of the latter is even suspected. The case herein recorded is of this type and is remarkable for the many diagnoses made during the period of observation by many competent clinicians and also because its true nature was finally revealed during surgical exploration.

The subject is a young woman, 25 years of age, whose birth and subsequent development were within normal limits. She was possibly somewhat precocious as a child, but remained well until March, 1915. At this time she noticed a severe pain in the right groin shooting into the flank; it lasted a half hour, but recurred every day, increasing in intensity. A few days later it assumed a girdle character. Prolonged rest in bed relieved the paroxysms, but when she got up three months later she became conscious of a constant pain over the appendix region and stabbing pains over the anterior surfaces of both legs from the knees down. Difficulty in the gait was noticed at this time; her legs felt weak, not stiff, and walking increased the pain in the side. A month later her disorder was diagnosticated gastric ulcer. She was treated for this, losing her pain, gaining weight and experiencing improvement of gait, although rapid walking still produced pain on the right side. She worked off and on, never regaining her strength, for one year. In August, 1916, she noticed a mass over the right side of the sacrum; this was opened and a profuse serosanguinolent discharge drained for ten to twelve days, the wound not healing for six weeks. While it was draining, she felt much stronger and could walk better. After the wound closed she experienced pain and stiffness of the back; a few weeks later another mass appeared at the site of the former and a gradually increasing difficulty in walking, characterized chiefly by a complete left and partial right drop-foot. There have been occasional cramps in the muscles of both calves, also casual difficulty of urination.

Physical Examination.—Examination in February, 1917, showed a waddling, steppage gait; double drop-foot, left complete, right partial; atrophy of both anterior tibial groups; otherwise the gross muscular power of

legs and thighs was well preserved. Knee-jerks and arm-jerks were absent; no Babinski; abdominals and epigastrics were present on the right side, absent on the left. There were rigidity and tenderness of the dorsolumbar spine, but no kyphos. Sensation was quite intact, except for a moderate hypesthesia over the dorsal surfaces of both feet. Otherwise physical examination was negative.

Diagnosis.—The diagnosis of compression myelitis probably from Pott's disease was made by Dr. Collins and the girl was referred to an orthopedic surgeon who concurred in the diagnosis and applied a plaster cast. She obtained relief from pain in the back, but otherwise remained in the same condition. As there had been some question about the roentgenographs of the spine, it was decided to remove the cast and submit her to an exploration. At this time the diagnosis of spina bifida occulta, cord tumor and syringomyelia were considered. Exploratory puncture of the cyst gave a pale, yellow fluid, containing albumin, but it did not reduce Fehling's solution. Fluid obtained by lumbar puncture gave normal fluid reaction.

Electrical examination showed reaction of degeneration in the anterior peroneal group and in the intrinsic muscles of the feet.

On March 22nd, exploratory laminectomy was done by Dr. Elsberg. The arches of the first, second, third and half of the fourth lumbar vertebræ and later of the twelfth, eleventh, tenth and ninth dorsal vertebræ were removed. The hemorrhage was small and there was no escape of cerebrospinal fluid. The cord was enormously swollen and filled up the entire intradural space. The arachnoid was everywhere firmly adherent to the pia, so that there was no subarachnoid space. With a probe, adhesions were felt at various levels and more arches

were removed in the search for the cause of the obstruction. Finally it was clear that the obstruction to the probe was due to the swollen cord. The surface of the cord was somewhat yellow and the veins slightly more prominent than normal. The cord itself felt cystic, and with an aspirating needle at least 30 c.c. of clear fluid were withdrawn. A small incision made into the cord at about the first lumbar level, about 1 centimeter in length, opened into an enormous hydromyelia cavity. The probe could be passed upwards inside the cord for at least 10 centimeters and downwards to the tip of the conus, so that the cavity was at least 20 centimeters in length and 1 centimeter in diameter. There was no doubt that the cavity was inside the cord.

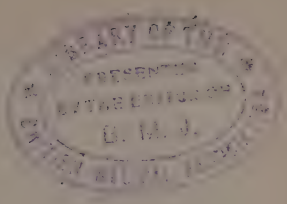
The small mass over the middle of the sacrum proved to be a little cyst in the subcutaneous tissue and was easily excised. The convalescence was characterized by a moderately elevated temperature lasting two weeks and paresis of the bladder. At first there was rather complete paralysis of motion of both legs. Sensation was undisturbed from the thighs down. There was loss of pain, tactile and thermal sensibilities of both legs, with exquisite tenderness of both calves. The bladder resumed its function at the end of a couple of weeks and the cutaneous sensory disturbance subsided; but the muscular tenderness increased to such an extent that contractures of the posttibial and ham-string muscles developed. Although they were later improved by passive exercises, they still persisted. The knee-jerks returned. The left abdominal and epigastric reflexes were now present, and there was decidedly more motion of dorsiflexion of the right foot and toes than before operation.

The origin of this cavity is extremely obscure. There are no positive evidences of congenital teratological le-

sions such as hydrocephalus or spina bifida, nor was it possible to demonstrate the existence of anything causing compression of the central canal. Exostoses, tumors, spondylitis and chronic hypertrophic meningitis could all be excluded, although objection to this might be raised because of the roentgenograph of the lumbar spine. The changes were so vague and indefinite, however, and there were so many different opinions as to the presence of actual changes, that one is not warranted in assuming that they really existed. Besides, the cavity extended so far above, as well as below this level that even if any definite changes in the dura had been discovered during operation it would be unjust to consider this as the cause.

The absence of objective sensory disturbances illustrated in a striking manner to what extent function may be retained in pathways subjected to great pressure, provided it be applied slowly and distributed evenly.

The cyst in the sacrum is also a puzzling factor. It seemed certain that there must be some connection with the cord lesion, because of the general improvement noted after its evacuation. At operation, however, it was shown to have no communication at all with the spinal canal, and hence could have exerted no direct pressure.



CHRONIC PRODUCTIVE LEPTOMENINGITIS

JOSEPH COLLINS, M.D.

A married woman of 53 was admitted to the Neurological Institute, July 25th, 1910. Her complaint was of pain and paresthesia. The former was chiefly in the neck, shoulder and hand of the left side; the latter, of the hands, feet, bust and buttock—sticking sensations in the hand, sensations as of strings tied around the toes, as of an elastic band stretched across the bust, as of sitting on an air cushion. The left upper extremity was immobile and she maintained that it hurt her to move it or to have it moved, and the left hand had the appearance of being swollen. Her suffering was apparently great and her complaint of it was constant.

These symptoms had been in existence eighteen months or more, but she had not been in good health for many years. Shortly after she married, in her 26th year, she bore a child, and after that had one miscarriage. There was reason for suspecting syphilis, but no history of it could be obtained.

When she was 36 years old she began to have attacks, described as follows:—She would experience a queer sensation all over, then the eyes would roll upward, the right foot would move backward and forward like a pendulum and gradually the right leg would draw up until the knee approximated the chin. The face would twitch and then she would lose consciousness. At the end of about a half hour she would vomit and soon after regain conscious-

ness. After such an attack she would feel prostrated. These attacks occurred with varied frequency, sometimes as often as one a day, and continued for upward of a year; then they ceased. She was said to be suffering from nervous prostration. After the attack ceased neither she nor her family noticed any particular change in her mental or emotional make-up, but she never considered herself well. She was not strong and often felt badly without specific complaint.

When she was 47 years old she was seized with agonizing pain in the back of the head which radiated to the top of the head. After it lasted several days she thinks she became unconscious and remained so 48 hours. There was no convulsion attending this attack. During the attack she fell out of bed and fractured a rib, it was said. The diagnosis made at that time was hysteria. Her recovery was slow.

When she was 51 years old, that is, two years before she entered the hospital, she first noticed that she could not walk securely, and particularly that the right lower extremity was losing its dexterity and strength. She also complained frequently of attacks of dizziness. She went to a sanitarium and remained there for upward of a year, and while there began to complain of the pain and paresthesia mentioned above. Two months before she entered the hospital, a physician whom she consulted told her that her right shoulder had been dislocated; he gave her chloroform and "set it." She stated that the pain had grown worse since then.

Physical Examination.—Examination revealed a woman 5 feet and 4 inches in height, weighing 162 pounds, who gave the impression of general bodily fixation or rigidity. Her gait was cautious, with short steps and a rather uncertain stride, as if the movement were painful.

She swayed slightly, with the feet together and eyes open. The left upper extremity was slightly flexed and held close to the side. There was distinct tremor of the right hand when the fingers were outstretched.

There was no disorder of the tendon-jerks or the superficial reflexes. The entire left upper extremity was markedly hyperesthetic and she complained of pain on slight pressure anywhere over this area. There was some tenderness on pressure over the right arm and shoulder, but it was not very profound. It was difficult to examine satisfactorily postural sensibility in this extremity, so bitterly did she complain of pain when the parts were moved. There was no astereognosis. The left hand had the appearance of being swollen and the skin was somewhat glossy, as was the skin of the entire left upper extremity.

Examination of the eye showed the pupils to be 4 mm. in diameter, slightly irregular and somewhat sluggish in their reaction to light. The mobility of the eyeballs outward was apparently limited in each eye, but the eyes were not convergent. There was nystagmus in extreme directions. Vision was 20/40 in the right eye and 20/70 in the left. The optic discs were pale, slightly blurred at the nasal margins, the arteries narrow, the veins of normal caliber, the fields showing a left homonymous hemianopsia for colors and for small test objects.

X-ray examination of the left upper extremity showed a marked atrophy of the scapula and humerus. The axillary border of the scapula seems to have been split longitudinally, and there had been a fracture through the neck of the scapula separating the glenoid and coracoid processes from the rest of the bone. The cervical and upper dorsal vertebræ showed no abnormalities. There was no indication of cervical rib.

Laboratory Findings.—The Wassermann examination

of the blood serum was weakly positive, Noguchi test negative.

The examination of the blood was as follows:

Hemoglobin 82 per cent, erythrocytes 4,380,000, leukocytes 16,000; differential count: neutrophiles 59 per cent, large lymphocytes 22 per cent, small lymphocytes 12 per cent, large mononuclear 2 per cent, transitional 8 per cent.

The examination of the urine showed chemically no abnormality, save a marked excess of indican. Microscopically it showed hyaline casts and cylindroids, some red and white blood cells and much calcium oxalate.

A month later the same conditions existed in an exaggerated form. The impression of every one in contact with the patient was that the pain was very great. It no longer confined itself to the left upper extremity, but extended throughout the whole left side of the body, below the neck, and it was necessary to administer narcotics. The paresthesiæ were very annoying. They varied from time to time, and the most insistent was the sensation of the tight band around the toes, forearm or the arm. At this date she related that before she came to the hospital the sensation of a tight band around the left arm was followed by the appearance of black and blue marks. The immobility of the left upper extremity continued unchanged. When one attempted to move this extremity she complained that it hurt her cruelly, but the extremity was not sensitive to pressure. The skin was hyperesthetic, but deep-seated pressure of the nerve trunks did not cause pain.

There was no disorder of the vascular system, and aside from hyaline casts, there was no constant abnormality in the urine. Pentose was searched for on many occasions but never found.

On October 3rd, that is, something more than two months after she came to the hospital, the patient, while in the act of turning off the electric light, lost consciousness and fell, striking her head and face. This was followed by severe nose bleed, badly swollen face about the eyes, and contusion of the scalp. The patient had no recollection of the incident; apparently there was no warning of the attack. The pain continued unabated, and from this day she began to experience pain also in the right upper extremity. It was of the same character, that is, of burning pain, but less intense.

Examination made three months after she came to the hospital showed some distinct evidence of the progression of the disease. She walked with very short steps, cautiously, with a zig-zag inclination. The appearance of general immobilization was even more striking than before. The head was held rigid, the left upper extremity was flexed at the elbow at an angle of about 100 degrees and adducted. Voluntary movements were limited to slight exertion of adduction and abduction of the arm, and even these movements were painful. The skin of the hand was glossy, the fingers were tapering and its entire appearance was one that is often seen after prolonged forced inactivity. Immediately above the left clavicle there was a soft mass which appeared to be of the size of the half of a hen's egg. It was impossible to tell whether it was adipose tissue (the patient was fat), it being so tender that thorough investigation of it was impossible. There was no real paralysis and no noteworthy alteration of the reflexes. The right plantar response was suspicious of extension, but this was finally interpreted as a defense movement.

Examination of the eyes on this date showed the same condition as on the previous examination, save that the

former hemianopsia for red with normal fields for white had now been replaced by loss of the left upper quadrant for white and for blue. For red and for green there was a loss of the entire left half of the field.

Examination made on the 16th of December, 1910, practically five months after the woman came into the hospital, revealed a condition of affairs similar to that of the first examination, only more advanced. The notes briefly recapitulated are as follows:—The expression of the face was that of a masque. The patient lay in bed, unable to arise without assistance; both forearms were flexed at right angles, the arms adducted. The fingers were tapering, separated and over-extended, in the left hand more conspicuously than in the right. She was unable to close either hand. She could not tell the nature of objects put into her hand or what was written on her palm. It was possible to move the fingers and hands, but such movements were extremely painful. All attempt at voluntary movement of the upper extremities was very painful. She stood without assistance but when she walked the steps were short, irregular, jerky; the head fixed. Surface temperature of the left foot was lower than that of the right. The skin over both lower extremities was glossy. There was no ataxia of the lower extremities and active and passive movements were ordinarily accomplished. The tendon-jerks were lively but not exaggerated. The cutaneous reflexes were normal. There was no clonus or Babinski phenomenon. Sensory examination showed an area of hyperalgesia as large as the hand, on the back of the neck, anesthesia of the fingers of the left hand and thermohypesthesia over the entire left half of the body from the clavicle downward, in front and behind. The examination of the eyes showed no particular change save that the hemianopsia had become more complete.

Briefly summarized the case was as follows:—A woman of 53 began to have, when she was 37 years old, attacks of convulsion—tonic spasm in the right side of the body followed by loss of consciousness, terminating in vomiting. These attacks were in existence about a year and a half. When she was 47 years old she had an attack of intense pain in the back of the head of several days' duration, after which she became unconscious for 48 hours. She was never strong after this attack and often complained of vertigo. When she was 51 she began to have pain and paresthesia in the left side of the body. The pain was of a burning character and the paresthesia was chiefly a compression. Immobility of the left upper extremity developed sequential to their existence. After these symptoms had been in existence on the left side of the body for about two years they developed on the right, the important antecedent having been an unheralded attack of loss of consciousness. The left upper extremity was immobile from pain; the right became so later. The skin had a glossy appearance and was at first hyperesthetic, then thermohypesthetic.

Diagnosis.—There were no physical signs of diagnostic importance save those referable to the eyes, a mild degree of optic nerve atrophy, possibly postneuritic, and left homonymous hemianopsia which had increased in intensity during the time the woman was under observation. The Wassermann test of the blood was not conclusive, but the attack of iritis, which she had when she was 33 years old and three years before the beginning of any of her symptoms, was suggestive of syphilis.

Two diagnoses were considered. The most plausible seemed to be chronic meningitis, spinal and basilar, probably of syphilitic origin. Such lesion might account for all of her symptoms, and if it were of a syphilitic nature, it

was thought to be quite possible that the iodid of potassium which she had received from various physicians might have influenced the course of the disease. The sluggish response of the pupils to light was also thought to support this diagnosis. The burning pain, the various paresthesiæ and the bodily immobilization could all be easily explained as evidences of encroachment upon the spinal roots by the meningitic process. Assuming that this disease of the meninges extended into the skull, the homonymous hemianopsia might be the result of implication of the optic fibers behind the chiasm. It would be quite possible also to explain the pallor of the optic discs by similar implication of the meninges over the quadrigeminal body, particularly the anterior. It was thought also that such diagnosis might account for the extraordinary concomitants of the "nervous prostration" of 17 years before. Assuming that the involvement of the meninges extended at that time to the Rolandic cortex, it would account for the convulsions and drawing up of the right leg, the unconsciousness, etc. The objection to such assumption would seem to be the cessation of such symptoms. It was realized that such cessation is not by any means uncommon in cerebral syphilis. The accession of illness which she had when she was 47, manifested by insupportable pain in the back of the head followed by unconsciousness for 48 hours, and this in turn by prostration and vertigo, was looked upon as further substantiation of this assumption.

We can find no record that a lumbar puncture was done. The lumbar puncture was not then a routine diagnostic measure, as it has since become. We know now that it would have assisted enormously in making the diagnosis.

The second diagnosis that was considered most probable was lesion of the optic thalamus, which was thought

of more seriously when hemithermoanesthesia was added to her other symptoms. Disease of the thalamus produces motor, sensory, secretory and emotional symptoms. Any of these may be absent. In fact, study has shown that lesions confined narrowly to the thalamus do not produce motor paralysis, but sensory symptoms, and particularly burning pain. Paresthesiæ and vasomotor disturbances have been shown to be dependent upon lesions of the basal portion of the thalamus. It was realized that rarely is any lesion, save tumor, confined narrowly to the thalamus, and it was difficult to explain the antecedent manifestations of this patient's attack on the supposition that the thalamus alone was the seat of the disease.

The patient's suffering increased to such a degree that it was decided early in January, 1911, to cut the posterior roots of the seventh and eighth cervical and first dorsal segments. When the laminectomy was done, it was found that the pia-arachnoid was so thick and adherent that it was scarcely possible to separate it from some of the roots before they could be sectioned. Small pieces of the pia were removed for microscopic examination. These pieces showed a pronounced connective tissue, hyperplasia, curly, thickened, connected tissue fibers with few nuclei. All vessels showed pronounced arteriosclerosis and typical obliterative endarteritis. In some places there was complete occlusion of the lumen of the vessels with proliferation of connective tissue in the media and also of the endothelium of the intima. In a few places considerable round cell infiltration was found in the perivascular tissues, but there was no coatsleeve infiltration. The smooth muscle cells of the media had undergone complete degeneration and appeared as granular detritus which took a yellow tint with the Van Gieson stain. There was

nothing in the appearance of the specimen to indicate the origin of the disease.

Subsequent History.—The patient made a good recovery from the operation, but we regret that she did not experience any relief either from this or from the administration of salvarsan, two doses of which were given intramuscularly, or from other antisyphilitic medication. As a matter of fact her suffering increased and she became gradually asthenic and died.

The following is a brief report of the changes in the central nervous system:

Report of the Microscopical Examination of the Sections

Central Nervous System:

Spinal cord:—Sections fixed in formalin. Stained with hematoxylin and eosin, with hematoxylin and acid fuchsin and by the Van Gieson method.

The nerve tracts are not altered; no degenerations apparent.

Very slight amount of brownish pigment in nearly all of the anterior horn cells throughout the cord. Otherwise the anterior horn cells are normal.

The pia throughout is markedly thickened. The thickening consists of an acellular substance in which the blood vessels are buried. The walls of some of the arterioles are somewhat thickened. There is no cellular infiltration around the blood vessels. The thickened pia encloses the nerve roots in a firm sheath where they leave the cord.

Cerebrum:—The material is fixed, some of it in formalin, and some in osmic acid. The sections were stained by Weigert's hematoxylin, hematoxylin, eosin and thionin.

The pia is apparently normal—no thickening or only very slight in some places.

The walls of the blood vessels are not appreciably thickened and there is no cellular infiltration about them.

The gray matter of the cortex is normal in appearance, no degeneration of nerve fibers being seen by either the Marchi or the Weigert methods.

Sections of Liver, Kidney and Spleen: Fixed in formalin, and the cut sections stained with hematoxylin and eosin, and with the Van Gieson stain.

Liver:—Advanced fatty degeneration. The fat is in large droplets resembling that seen in alcoholic fatty degeneration of the liver.

The portal canals are normal.

Kidney:—Acute degeneration (coagulation necrosis) of the proximal renal tubules. No interstitial changes. Glomeruli are normal. Coagulated exudate in the spaces between the glomeruli and their capsules and within the kidney tubules.

Spleen:—Many areas of hemorrhage composed of slightly altered blood. Small amount of brown pigment throughout the section.

Anatomical Diagnosis:

Chronic productive leptomeningitis of the spinal cord.
Advanced fatty degeneration of the liver.
Acute degeneration of the kidneys.

DISSEMINATED LESIONS OF THE SPINAL CORD DUE TO MALARIA

C. BURNS CRAIG, M.D.

The virulence of malarial toxin has never been adequately appreciated. In our desire to forget the days of superstitious credulity when all disorders attended by fever were "malaria," we have neglected to estimate carefully the potentiality of the plasmodia to cause organic disease of the nervous system.

The protean symptomatic display of another chronic infection, syphilis, is widely granted. It is probable that too frequently the presence of chronic malaria is overlooked in the search of an explanation of symptoms. Particularly is this true in the cases of the immigrants of recent years who come from malarial infested regions.

Ever since the periodicity of the chill and fever in plasmodial infections began to be recognized as a definite entity, unlike other fevers, the tendency to neurological manifestations has been marked. Maillot, in 1836, wrote: "In the midst of the works on intermittent fevers which succeed one another, is the predominating idea which associates intermittent fever with lesion of the nervous system." He also records the first neurological case due to malaria. It was characterized by successive paralysis of the right arm, the left arm, the lower extremities, the vesical sphincter and respiration. There was a dearth of contributions to the subject until 1851, when a young military medical officer, Ouradou, serving in Algiers, wrote

his university thesis on "The Paralyses in Intermittent Fever," in which he refers to the case of Maillot and describes five cases of his own which displayed prominent neurological symptoms. Two of these resulted in permanent paraplegia.

Since that time, there has been an increasing number of cases, reported from all lands, showing various paralyses and disseminated lesions in the nervous system due to malarial infection. In the following case, an individual, subject to chronic malarial infection, developed a group of symptoms pointing to disseminated lesions of the spinal cord, and it is quite legitimate, especially in the absence of other causative factors, to attribute them to that infection.

The patient, a Sicilian 42 years old, has been in this country eight years. He was quite well until one year ago when the left leg began to grow weak and he had pain in the left side of the small of the back. After a month he began to experience pain and itching in the right thigh, and a sensation of retraction of the leg of that side. At the same time he became severely constipated and for a few weeks was occasionally incontinent of urine. There was no impairment of potency.

About nine months ago he noticed that cold things felt warm to the right lower extremity. For example, when sitting on the water closet it felt cold to the left buttock and warm to the right, or when in a cold wind, the left leg would feel cold and the right warm. During the past year the lower extremities have gradually become stiff so that his toes scraped in walking.

Of great importance in his past history is the fact that for six years previous to his coming to this country he suffered each year, about the month of August, with chills and fever, occurring intermittently and lasting ten days

or two weeks. He treated this illness similarly each year, namely, with purging and quinin.

Physical Examination.—Examination showed the following evidences of pyramidal tract disorder,—a stiff-legged spastic gait, increased knee and ankle jerks, greater on the left side, patellar and ankle clonus on both sides and a double Babinski sign as well as absent epigastric, abdominal and cremasterics. The so-called Hoffman sign was present on both sides. The sensory changes consisted of diminished tactile and pain sensation of the right lower extremities and dissociation of temperature sense. He invariably interpreted cold as warm.

The pupils were slightly unequal, the right being larger and their contour not quite circular, but the reaction to light and accommodation was immediate. There was a slight grayish cast to both optic discs. There was also slight nystagmus, more pronounced of late on looking to the right. The spleen was enlarged and descended about 6 cm. below the costal margin on inspiration. Its border was smooth and slightly rounded. The skin was of a curious yellowish cast.

Laboratory Findings.—There was no anemia, as the hemoglobin was 90 per cent and the red cells 4,900,000. The Wassermann reaction was negative in the serum and spinal fluid. There was no excess globulin in the spinal fluid.

On the 10th of December he was given 10 grains quinin. On the morning of the 11th he received 5 grains. About noon he had a chill and his temperature rose from 97 degrees to 101 degrees, but it was again 97 degrees the following morning.

Diligent search for plasmodia was made before and after the chill, but they were not found. On December

31st he was given 15 grains quinin intravenously. About 1½ hours later he had another chill. The blood was again searched before and after the chill for plasmodia, without positive findings.

Diagnosis.—The enlarged spleen, the history of typical malarial attacks and the characteristic reaction of chronic malaria to quinin, are sufficient warrant for attributing the lesions of the central nervous system to malarial infection.

SUDDEN PARAPLEGIA OCCURRING AFTER INJECTIONS OF AUTOLYSIN

JOSEPH COLLINS, M.D.

It is often difficult and sometimes impossible to make a satisfying diagnosis of the nature of the disease which is causing definite and well marked spinal cord symptoms, such as those of transverse lesion giving rise to partial or complete interruption of continuity of the cord. At the present time we have in the hospital a case which substantiates this statement so emphatically that I have decided to present him even though the diagnosis of the nature of the lesion cannot be affirmed.

The patient is a surgeon, 65 years old, who came to New York a few weeks ago to be treated for cancer of the bladder, by the autolysin that is spoken of as Dr. Beebe's serum. He had received the twentieth injection on February 2nd, 1917. When he awakened on February 3rd he was paraplegic: complete motor and sensory paralysis existed with incontinence of feces and loss of expulsive power of the bladder. I saw him four days later; he was able to flex slightly the left lower extremity at the knee. Neither the tendon-jerks of the lower extremities nor the skin reflexes could be elicited. All forms of sensibility were disordered below a line drawn around the body two inches above the nipples. In the feet and legs tactile, thermal and pain stimuli were not perceived. As the stimulus was approached to the trunk there was faint perception of it, and gradually it became more percept-

ible until the 4th intercostal space was reached, where it produced normal reaction. Day by day since, the motor and sensory paralysis have become less intense, and he is now able to move the lower extremities, the left with much freedom and display of strength, and the right to a considerable degree. He is able to turn over in bed and to sit in a chair. The sensory disturbance has decreased *pari passu.* Touch, pain and thermal stimuli are perceived indifferently below the line of demarcation, and the further one goes from it the more distinct is the sensory disturbance. While the sensory disturbances have receded, they have been distinctly more marked on the left side of the body; this side also displays the lesser motor disturbance. In other words, the paralysis tends in its retrogression to be of the Brown-Séquard type. As the motor power returned the tendon jerks of the lower extremities became elicitable, and now they are plus on the right side and about normal on the left. The Babinski big toe phenomena and exhaustible ankle clonus are likewise elicitable in the right foot. The condition at the present time, therefore, is that he has a sensory motor paraplegia with tendency to Brown-Séquard distribution, with the customary ancillary phenomena all in recession.

The noteworthy features of the patient's history are: When he was 33 years old he went to South America and while there contracted malarial fever. One day he became exhausted and greatly overheated from his efforts in extinguishing a fire. He retired that night greatly fatigued and awoke the next morning with paralysis of the lower extremities. He was not absolutely paraplegic, but he had to remain in bed for several weeks. He made a complete recovery, but for six months he had some difficulty in walking upgrade. When he was 46 years old he

contracted syphilis while operating, the local manifestation of the infection being on the left index finger. He delayed treatment until the rash appeared, but then took protiodid of mercury for two and one-half years. Soon after this infection he experienced weakness of the legs, and although he did not become paraplegic, he had distinct enfeeblement which lasted several weeks. When he was 50 he had a mild albuminuria, for which no cause could be found, which lasted two or three months. A similar condition had occurred when he was 35 years old. At 51 he developed, after financial reverses, a profound neurasthenic state: insomnia, depression, indecision, unrest, introspection, disordered perspective and physical debility associated with indigestion and nocturnal urination. In addition to this he complained of pain in the shoulder blades, a general ache, frequent backache like lumbago, a sense of soreness and lameness in the outer side of the left leg, of pain in the ears. When I saw him at that time, his blood pressure was low, 130, the pulse rapid, averaging about 100, the heart sounds feeble, particularly the first. The second sound was not accentuated. The face was red, of a deep hue, almost cyanotic, and white areas remained for some time after pressure. The gastric juice contained no free hydrochloric acid and the colon was distended. There was marked coarse tremor of the hands which increased on intention. The pupils and tendon-jerks were normal. There was no tremor of the face. Examination of the urine and feces was negative. The diagnosis of neurasthenia was made, the underlying factor being a vasomotor disequilibrium. He recovered in about a year under general tonic treatment. When he was 54 a Wassermann examination of the blood showed it to be positive; the spinal fluid was negative. He was under treatment (sal-

varsan and mercury) about two years. Repeated applications of the test after that time always gave a negative reaction.

He remained in fairly good health until his sixtieth year when he began to have symptoms of vesical irritation, delay in starting the urinary stream, imperative urination, occasional slight incontinence and pain of varying intensity. After a year's treatment a suprapubic prostatectomy was done, the middle lobe of the gland being removed. The pathologist reported that the tissue removed showed no indication of malignancy. For the year following the operation he was comparatively free from symptoms. At about this time he began to have paresthesia of the feet, and rather indefinite pains in different parts of the body, a sensation of lameness in the front of the thighs and of stiffness in the hips and back on arising after he had been sitting for any length of time. He became fearful lest the luetic infection was still active, but the examination of the blood serum and the cerebrospinal fluid was entirely negative; nevertheless, he took three or four injections of salvarsan, one of them intraspinous. The symptoms of what were called rheumatism, stiffness of the neck and pain and stiffness in the joints, disappeared after several carious teeth were removed. Nevertheless, there were some symptoms of a sensory nature which persisted. One of these was a burning sensation around the chest, beneath the right nipple; another, a disagreeable, stiff, painful sensation down the outer side of the thighs. Neither of these was continuous. The first began as a pain around the heart, then it seemed to move to the right side; it would come on in attacks, the attack lasting a variable time, from an hour to a day. He consulted a great many physicians and was looked upon as a syphilophobiac. The symptoms of

cystitis, in the meantime, gradually increased so that three years after the operation he was having as many and as severe symptoms as he had had before he submitted himself to operation. In May, 1916, he consulted a colleague, who told him, after careful examination, that the lesion responsible for the cystitis was probably cancer of the prostate. In July, 1916, this opinion was corroborated at the Mayo Clinic, and he was advised to submit himself to radium treatment. Instead of doing so, he returned home and received radium treatment in the urethra for a few weeks. In August, 1916, he went to Chicago and subjected himself to the treatment known as mesothorium. In the late autumn of 1916, he came East and consulted many physicians in Boston, and so far as he can tell most of them concurred in the diagnosis of malignancy. Finally he yielded to the advice that he take the serum prepared by Dr. S. P. Beebe, and he had received twenty injections when his present trouble developed. The attack came on in the night. He had not noted on the previous day that his legs were any more infirm than they had been for some time past, nor had he complained of pain in the legs.

A brief recapitulation of this case therefore seems to be as follows: (1) Malarial infection when thirty-three, partial paraplegia after strenuous effort at this time, *i.e.,* while the infection was in the blood; recovery in six months.

(2) When thirty-five albuminuria lasting some months; recurrence again for a few months at fifty.

(3) Syphilitic infection when forty-six. Apparent recovery under vigorous mercurial treatment. Four or five years after infection he noted uncertainty in walking; feet felt as if they were wrapped in cotton.

(4) When forty-nine years old an attack of neurasthe-

nia with profound vasomotor symptoms. Complete recovery within eighteen months.

(5) When fifty-five the serum Wassermann was found to be positive, the cerebrospinal fluid negative. The serum became negative after he took treatment, and it has remained so.

(6) In his sixtieth year he developed cystitis and the middle lobe of the prostate was removed one year later. Amelioration of symptoms for upward of a year.

(7) When sixty-one years, paresthesia of the different parts of the body, feet, outside of the thighs, accompanied by stiffness of the legs, developed. These paresthesiæ were inconstant and of varying intensity. Two years later they included a burning sensation around the right nipple, coming on in paroxysms.

(8) When sixty-four the diagnosis of carcinoma of the prostate was made and he was subjected to radium emanations indifferently employed.

(9) When sixty-five, while he was receiving a cancer autolysin there developed sudden paraplegia with symptoms and signs indicating a complete transverse lesion of the spinal cord in the upper dorsal region.

(10) Gradual amelioration of the symptoms indicative of such a transverse lesion.

Physical Examination.—This case presents unusual difficulties of interpretation. The physical examination reveals, first, thickening of the floor of the bladder, which is not nodular. Dr. J. B. Squier, prevented from making cystoscopic examination because of the profound cystitis, is unable to say whether it is a chronic hypertrophic process or a carcinoma. Second, the *x*-ray examination shows in the fifth, sixth, and seventh cervical vertebræ a certain degree of patchiness due to the absorption of the lime salts, which Dr. Imboden considers to be sug-

gestive of metastatic carcinoma. Third, a partial sensomotor paraplegia which is the sequelæ of the complete sensomotor paraplegia, the line of sensory demarcation being two inches above the nipple in front and just below the nipple line behind. Fourth, normal cerebrospinal fluid and a negative Wassermann reaction of the blood serum. Fifth, no indications of disease of the spinal column. Sixth, dilatation of the aorta.

Diagnosis.—The condition that we are dealing with here may be one of three: The patient may have a carcinoma of the neck of the bladder with a metastasis in the lower cervical and upper dorsal vertebræ, which is producing compression of the cord. The metastasis may be in the substance of the cord, and a hemorrhage in such metastasis may have produced the suddenly developing symptoms of transverse myelitis; the hemorrhage subsiding may possibly have allowed that degree of reparation to go on which must have gone on, in order to display the degree of recovery which he has already made. I am told that one of the immediate results of exhibiting the autolysin is to cause a hyperemic state of the neoplastic formations which it is desired to influence. The chief objection to this assumption is that previous to a fortnight ago, this patient had no symptoms of disorder of his spinal cord, and particularly of such encroachment upon his spinal cord as a tumor would cause. Unless it be assumed that the paresthesia which he has had for seven or eight years past were the indication of such disease, the patient was in truth free from all spinal cord symptoms until he was seized with a stroke. It is quite possible to conceive of a metastasis in the spinal column itself which might exist for a long time without producing other symptoms than those of indefinite irritation of the spinal cord and its covers. In such a case,

however, there must almost of necessity be indications of disorder of a function of the spinal column—rigidity, limitation of movement, muscular spasm, pain of varying intensity but referable to the seat of disease, tenderness, deformity, and so forth. In this case there were none of these. If we assume that his symptoms, due to interruption of the conductivity of the spinal cord, were due to tumor, the incidence of the stroke must have been the result of a hemorrhage in such tumor. The invincible fact against such assumption is the rapidity with which these symptoms are disappearing.

Second: The lesion may be a transverse myelitis of septic origin. Experience teaches us that the spinal cord is particularly susceptible to secondary infection from septic process in the genito-urinary tract. This patient has had such an infection for a considerable number of years, and it is not beyond possibility that the spinal cord lesion upon which the paralysis is dependent, is a transverse myelitis whose origin was in a septic inflammatory process of the deep urethra and bladder. Again, the chief objection to such assumption is the rigidity with which these symptoms are associated. Unless the lesion were of the nature of a septic thrombosis, which, when it occurred, was followed by an edema whose pressure caused a condition of affairs tantamount to interruption of continuity of the cord, the rapidity and degree of recovery which the patient has made, added to the absence of trophic manifestations, makes it very improbable that the lesion is a transverse myelitis.

Third: It is quite possible, however, that the lesion in this case is a myelomalacia of vascular origin incident to profoundly altered arterial tonus in the upper dorsal cord. It is very suggestive that the patient developed a paraplegia after his first protozoal infection and it is like-

wise interesting to note that soon after the second protozoal infection he had marked weakness of the lower extremities, sensory and motor symptoms. This would certainly indicate a low resistant power of his spinal cord. This resistant power has been steadily lowered by the experience of the past five years, and when the sudden effects of the autolysin, whatever they may be, manifested themselves, it is quite possible that such vascular atonia occurred at such a definite level of the cord as to produce the equivalent of thrombosis with resulting stasis and sequential edema. When vigorous restorative measures and rest were utilized he began to recover from the effects of such transverse lesion, and should recovery go on to such a degree as to permit him to walk, I should have no hesitancy in accepting this explanation of his symptoms.

On the other hand, I cannot, however, wholly eliminate the suspicion that syphilis may, despite the negative state of his blood and cerebrospinal fluid, be at the bottom of his present predicament. The belief has been forced upon me that one who contracts syphilis in the years of his late maturity never wholly recovers. It is by no means improbable that there is a syphilitic endarteritis in certain branches of the anterior or posterior spinal artery. I need scarcely say that such lesions can and do exist without evidence of syphilitic disease in the blood or cerebrospinal fluid.

The advanced structural disease of the aorta revealed by the *x*-ray and corroborated by the physical examination bespeak profound syphilitic disease of the arterial system. Indeed it is quite possible that the thoracic symptoms that he has had off and on for the past three or four years had their origin in disease of the aorta. Likewise, the paresthesia of the lower extremities may be explained

by circulatory disturbance in the spinal cord incident to syphilitic endarteritis. The paraplegia may have been the result of a thrombotic obliteration of some of the spinal cord blood vessels in the upper dorsal cord, and it and the resulting edema may have been responsible for the symptoms of complete interruption of continuity of the cord. The subsidence of the edema was followed by disappearance of such manifestations of interrupted continuity and the permanent symptoms of myelitis which he is now showing would be the indication of the permanent lesion of the cord due to the thrombosis.

It may seem to you quite unnecessary that such interpretation as I am inclined to put upon his spinal cord symptoms are justified, in view of the fact that he has a malignant disease of the prostate which not infrequently gives rise to metastasis in different parts of the body, including the vertebræ. But as was said before, I can see no good reason for looking upon the spinal cord symptoms in this case as being dependent upon disease of the spine, and moreover, neither Dr. Jerome M. Lynch, our consulting enterologist, nor Dr. J. B. Squier has been willing to say he has carcinoma of the bladder.

ATAXIC PARAPLEGIA—COMBINED SCLEROSIS

JOSEPH COLLINS, M.D.

The pathogenesis of combined sclerosis is as obscure as almost any problem in the realm of neurology. Aside from its occurrence, its probable dependency upon disordered states of the blood, such as profound anemia and certain hemic intoxications, we know practically nothing definite about it. The brief contribution, which we desire here to make, may throw no light upon this obscure question, but we desire to point out that in a case which has been for a long time under our observation there was found on examination of the cord an amount of arterial sclerosis which was not in keeping with the patient's years, nor had she been ill a sufficiently long time for one to say legitimately that it may have been incidental to the disease.

The history of the patient is briefly: A widow, forty-three years old, had when she was fourteen years of age a severe attack of scarlet fever, followed by such weakness that she did not get about for six or eight months. Aside from this she had always been quite well until the beginning of her present illness, which she dated from the winter of 1904. The initial symptom was paresthesia of the lower extremities, creeping, pricking, cold sensations extending from the waistline to the feet. These were not of uniform severity, but at the beginning of variable intensity. They soon absorbed her attention so completely that she had to give up work. In June, 1904,

i.e., five or six months after the onset of the paresthesia, she complained of stiffness of the knees, difficulty in walking, particularly in going up or downstairs, and occasionally trembling of the legs. The rigidity of the lower extremities increased rapidly and a month after its onset she could scarcely walk. About this time she first complained of cramps in the legs and of involuntary drawing up of the legs at night, of such severity that she would have to get up and sit in a chair for a while. There was no disturbance of the function of the bladder, such as slowness in starting the urinary stream, or hurried action, no complaint of vision or disorder of function of any of the cranial nerves.

Examination showed the common associated conditions of spastic paraplegia: all the tendon-jerks exaggerated, double ankle clonus, double Babinski, marked passive rigidity of both legs, no atrophy of the legs, abdomen held rigid, abdominal reflexes present at first, gone afterward, no objective sensory changes and no disturbance of deep sensibility. There was no nystagmus, ptosis, strabismus or disorder of the pupils. Psychro-esthesia was the principal subjective sensation, and upon the outer side of the thighs she said this sensation was most disagreeable.

Physical Examination.—Examination on February 8, 1906, showed practically the same condition as that indicated above. It is particularly to be remarked that no objective sensory disturbance could be made out, and the deep sensibility was normal. The spasticity had become much more pronounced. The abdominal jerks could no longer be elicited, possibly because of the marked rigidity and tympanitic condition of the abdomen. The upper extremities revealed no spasticity, but the direct myotatic irritability, especially of the supinators and of

the triceps, was profoundly increased. There was no nystagmus, no pallor of the optic discs, and the pupils were normal. From this time onward the spasticity increased; soon both legs became firmly flexed on the thighs; she became incontinent of urine, and bedsores developed. She had had a bedsore in the late fall of 1905, which healed rapidly.

Laboratory Findings.—Examination of the blood made on February 14, 1906, showed 3,000,000 red blood cells, 5,000 white, hemoglobin 65 per cent, color index normal, differential count normal. Previous examinations of the urine and blood had showed no abnormalities.

Clinical Summary.—A woman at the beginning of middle life developed symptoms of involvement of the posterior and lateral columns of the spinal cord. Clinically, the picture was one of spastic paraplegia with all its concomitants. There was nothing in the history of the patient to account for the disease. Examination of the blood showed finally a progressive secondary anemia. The condition of the patient did not improve and death followed.

Pathological Findings.—Examination of sections from the upper dorsal segment showed symmetrical degeneration in both crossed pyramidal tracts, the direct cerebellar and the posterior columns. Marchi degeneration of the same tracts was not found, although here and there, there were a few degenerated fibers. The degeneration at this level did not involve the root zones. The borders of the pyramidal tracts and of the portion between the root zones and the posterior columns showed a remarkable dilatation of the spaces occupied by the fibers. Van Gieson and Bohmer eosin stain showed the vessels everywhere in the affected portions to be in an advanced state of arterial fibrosis, *i. e.*, thickening of the media and adventitia,

with slight perivascular round-cell infiltration and swelling of the perivascular spaces. In many places there was seen a peculiar meshwork of connective tissue fibers and cells in the perivascular spaces, which gave the impression of having been caused by pressure from transudation of serum. Busch preparations showed a well-defined fatty degeneration of the vessel walls. The small capillaries were also much thickened, and here and there small petechial hemorrhages into the perivascular spaces or surrounding nervous tissue were seen. The elastic coat was in many instances split up into fine strands.

In the middorsal region where the degeneration resembled that in the upper segments, similar vascular changes were more pronounced. The dilatation of the perivascular spaces was more marked; there were a great number of thickened capillaries and more pronounced fatty degeneration of the vessel walls. At this level there was seen a peculiar dilatation of the central canal accompanied by thickening and some slight rarefaction of the walls. The dilatation was symmetrical, almost circular, but lower down it became elongated laterally, so that it appeared to be nearly as wide as the posterior columns.

In the lower dorsal regions, between the tenth and twelfth segments, a change in the distribution of the sclerotic vessels was seen. The changes in the vessels of the posterior and pyramidal tracts were still very marked, and the vessels of the gray matter also showed much greater involvement than higher up. The central canal was slightly elongated in the anteroposterior diameter; its walls were thicker, and the epithelium was better preserved.

In the midlumbar region, the variation in the dilatation of the central canal was even more marked. Here the en-

largement was almost complete along the anteroposterior diameter, while its walls were enormously thickened, often appearing quite edematous. Nowhere could an invasion of the horns be observed. At this level the arteriofibrosis in the gray matter was even more marked. There were several punctate hemorrhages, and the center of the anterior horns showed a softening process with körchen cells, eosinophiles and proliferating neuroglia cells. This softened area extended downward to the sacral portion and there gradually disappeared. The degeneration in the posterior columns remained unchanged. The oval field of Flechsig and triangular area of Gombault and Phillippe, as well as the entire root zone, were well preserved.

The vessels showed the same peculiar fatty degeneration and the characteristic thickening of the smaller arterioles and capillaries.

At the level of the third sacral the posterior columns showed practically no degeneration. The vessels were not nearly so thickened and showed very few changes as compared with those higher up.

The pia was evenly thickened throughout the entire length of the cord. The vessels showed everywhere moderate thickening, and an occasional obliterating endarteritis, which in one instance had gone on to complete transformation into connective tissue.

The medulla and pons gave a strikingly different picture. There was no degeneration to be seen in the pyramidal tracts either by Weigert or Marchi method. The pyramids throughout the crura, pons and medulla gave a constant and deep stain by the Weigert method. The vessels also showed much less involvement. While they were very plainly thickened and sclerosed, still the contrast with those of the cord, more especially in regard

to the smaller arterioles and capillaries, was very marked. There were no patches of softening. The perivascular spaces were moderately dilated and the vessel walls showed a small amount of fat, not at all comparable to those of the cord.

Unfortunately the brain was opened by the Virchow method and rather mangled. The pia was moderately thickened throughout and showed many small punctate hemorrhages which did not dip into the cortex. The vessels of the cortex were engorged with blood and showed a fairly well advanced state of arteriosclerosis. A few of the smaller meningeal vessels were blocked by an obliterating endarteritis. The head of the left caudate nucleus was studded with small punctate hemorrhages, which were apparently the result of transudation from the vessel walls, as in no instance were they seen very far from the vessels from which they arose. Neither the Busch nor Weigert method showed degeneration of the left internal capsule. The right lenticular nucleus showed also a few hemorrhagic conditions similar to the left side, and the right internal capsule showed no degeneration of its fibers.

Summary.—There were symmetrical degenerations of the crossed pyramidal tracts, direct cerebellar tracts and posterior columns, throughout the cord, with practically no involvement of the root zone or posterior roots and cells. While here and there there was pallor and a lack of staining properties of some of the roots, still when compared with the root zone there was practically no degeneration. The degeneration was confined solely to the cord.

The vascular changes throughout the entire cord consisted of marked thickening of the media and adventitia, with practically no obliterating endarteritis. Certain lo-

calized areas of the brain, and also the cerebral meninges, showed similar changes, but the brain substance itself as a whole showed no such changes as were found in the cord. These sclerotic changes were particularly marked in the degenerated areas of the cord, and it may be said that wherever there is degeneration, either in the tracts or in the lumbar region, where there is actual softening in the gray matter, the vascular changes are most profound. In other words, there is a typical picture of combined sclerosis accompanied by intense vascular changes.

Unfortunately, due to improper technique during its removal, the portion between the lower border of the olives and the eighth cervical segment was lost, so that it is impossible to state positively whether the changes in the pyramidal tracts were due to an area of softening at the decussation of the pyramids.

At first glance it might seem as though these vascular changes were simply coincidental with the evident secondary sclerosis of the glia, but while undoubtedly a certain amount of the sclerosis was due to this, still this same intense sclerosis, although not quite so marked, was found in the other portions of the cord, and therefore it does not seem as though they were simply the direct consequence of the degeneration.

Diagnosis.—While I do not ascribe positively these degenerations to the vascular changes, at the same time we have a very significant picture, especially in the middorsal region, where we have degenerated tracts surrounded by the dilated spaces for the fibers, which interpose themselves as a sort of barrier between the actual sclerosis and the healthy portions. In other words, we have the same picture as that which we get upon the periphery of cords showing senile changes.

The main objection to this hypothesis is that we have here the typical picture of a system degeneration, and that it is difficult to understand how or why this process should pick out these systems and spare the others. That it does not conform to the typical picture of arterial sclerosis with the patchy degenerations and multiple areas of softening, we are well aware, and with reason, since we find none of the thrombi and hemorrhages which occasion them. The changes denote rather a nutritional loss which has attacked the less resisting systems first, and those which may have been started, as in the crossed pyramidal tracts, by an area of softening in the region of the decussation. In these cases it is often impossible to differentiate pure systemic degenerations from these conditions, and of course the same etiological factor that caused the vascular changes may have occasioned the system degenerations. In that case, however, we should expect to find it at least in the lower levels involving the roots and root zones as in tabes. As a matter of fact experience has convinced me that the poisons that cause the lethal anemia, to which the combined sclerosis of these cases is sequential, often cause simultaneously widespread vascular disease.

AMYOTROPHIC LATERAL SCLEROSIS

C. BURNS CRAIG, M.D.

The patient is a German, born in Portugal, 41 years of age, and married. He is the father of four children. He denies specific infection and his only illnesses have been pneumonia when fifteen and a severe attack of sore throat four years ago. As a child he had throat trouble. At twelve years of age he suffered an insignificant trauma, being hit on the head by a piece of slate. He came to the Neurological Institute on January 11, 1917, complaining of inability to raise his arms; in fact, complete disability in the use of his upper extremities, disorder of speech, and difficulty in swallowing.

The present illness began 18 months ago with a mild numbness of the right shoulder. Following close upon this, he noticed that he was awkward with a pencil in writing, in his occupation as a shipping clerk. The slight disability in the use of the hand was succeeded by a heaviness of the entire right upper extremity. During the succeeding three months the right arm became progressively weak, so that at the end of that period he was unable to raise it, although he could still grasp a pencil after a fashion.

About this time he experienced difficulty in pronouncing certain words and frequent dysphagia. Six months after the onset in the right arm, the left arm became involved and passed through the same sequence of disabilities. The disorder in both upper extremities and

difficulty in speech progressed slowly but steadily. For six months he has been unable to write. Speech has become almost unintelligible and swallowing more difficult, fluid sometimes regurgitating through the nose.

Physical Examination.—The patient presents the picture of an advanced amyotrophy. He is a man of short stature—five feet three inches—and small bones. There is a pronounced atrophy of the muscles of the shoulder girdle, especially of the supra- and intraspinati, the deltoids and pectorals. The biceps and triceps, the muscles of the forearms, the interossei, the thenar and hypothenar eminences, are also involved in the atrophy. The atrophy is accompanied by loss of tendon and periosteal reflexes. In contrast to the wasted state of the upper extremities is the plump, rounded condition of the thighs and calves, which are feminine in form. The tendon-jerks at the knees are slightly exaggerated, but equal. The ankle-jerks are present. There is no Babinski sign nor clonus. The abdominal reflexes and cremasterics are present; the epigastrics are not obtained. The pupils are equal, round and react promptly to the light and in accommodation. Vision, hearing, taste and smell are not disordered. The tongue is small, atrophic, and the mucous membrane lies in folds upon it. He cannot protrude it beyond the teeth, and attempts at lateral movements are accompanied by exaggerated movements of the jaw; in fact, he cannot stick his tongue into his cheek; the palate hangs stationary. The voice is pronouncedly nasal and swallowing is decidedly difficult. Weakness of the sternocleidomastoids is demonstrated by having the patient erect his head from the retracted position. When he attempts to bring the head forward, a slow, steady pull of the sternocleidomastoids accompanied by contraction of the platysma myoides lasting four

or five seconds and ending with a final snap, brings it to an erect posture.

Diagnosis.—The patient is a classical example of amyotrophic lateral sclerosis, that is, he combines the three cardinal clinical pictures, that of chronic anterior poliomyelitis, bulbar paralysis, and spastic spinal paralysis. The only feature that is not particularly developed is the latter. The spasticity is very slight, nor do many of the objective manifestations of this condition exist. The absence of such phenomena makes it necessary, possibly, to distinguish this case from amyotrophic lateral sclerosis and to designate it as chronic poliomyelitis, spinal and bulbar. If during the progress of the disease fibrillary twitching of the atrophic muscles does not become more conspicuous than it is now and should the spastic phenomena fail to develop we should be obliged to make the latter diagnosis.

In this case syringomyelia, syringobulbia and gliosis can be excluded because of the absence of any sensory symptoms. Multiple sclerosis can also be excluded because of the absence of any of the cardinal symptoms of that disease. Experience has taught us that syphilitic process of the cord and meninges can cause symptoms very closely resembling those which this man presents, but in this case that can be excluded by the absence of a history of syphilis and for the reason that the blood and cerebrospinal fluid do not show any indications of syphilis. Were the symptoms in this case limited to the muscles supplied by the motor nerves arising from the medulla oblongata it need scarcely be said that this man would be a typical case of progressive bulbar paralysis. The speech disturbance, the dysarthria, the dysphagia, the disorder of phonation, the toneless cough, the atrophy of the muscles of the tongue and lips, and the lar-

yngoscopical examination are all typical of bulbar paralysis, but the profound and extensive atrophy of the upper extremities bespeaks the extensive involvement of the anterior horn cells of the entire cervical cord.

DISSEMINATED SCLEROSIS OR HEMORRHAGE INTO A SYRINGAL CAVITY

JOSEPH COLLINS, M.D.

It is difficult to realize that disseminated sclerosis has been recognized generally, only during the past few years. The history of the disease in this country is really an extraordinary chapter. Even to-day it is difficult to convince many physicians that the diagnosis is rarely predicated upon the triad of symptoms,—intention tremor, staccato speech and nystagmus which Charcot first pointed out were so characteristic of the disease. There can be no doubt that next to the syphilitic diseases of the central nervous system, disseminated sclerosis is by all means the most important. It displays itself in so many bizarre ways; it apes so many other organic diseases of the spinal cord particularly, such as different forms of myelitis; it comports itself so differently in different instances that it is small wonder that the practitioner finds no satisfaction in hazarding a diagnosis of cases which do not conform to the so-called typical display.

The case herewith described is, I believe, one of disseminated sclerosis, but it may quite well be an example of hemorrhage into a syringal cavity, a condition, I need scarcely say to you, which is very unusual.

A young man, 29 years old, a draughtsman, was well, so far as he knows, until about the 4th of July, 1917. At the age of fifteen he had an attack of typhoid fever.

On the 4th of July he was working in Pittsburgh at his profession when he noticed that his right upper and lower extremities were getting so weak that he had to discontinue work. At the same time he remarked that the other side of the body was the seat of a tingling sensation-numbness, which gave him a desire to rub his hands and forearm and to flex his fingers.

He went to a physician, who found that he had a mild fever of about 100°, which continued for two days; the patient was then seized with a severe cutting pain in the right shoulder. This pain lasted about an hour, following which, he remarked that his right upper extremity was much weaker than before the pain came on. The right leg, however, did not seem to have had this weakness increased by the occurrence of this pain.

At this time, namely the 7th of July, he had some difficulty in starting the urinary stream.

He came to see me on the 22nd of July. He walked with a slight limp of the right leg; still he walked up the steps and into my house unaided, dragging his right leg somewhat, the right hand being held in a position of slight flexion. In addition to this complaint, he had what he termed a peculiar numbness of the left side of the body from shoulder to toes. Aside from that, there was no complaint, save of weakness.

Physical Examination.—His examination then showed that there was a typical Brown-Séquard paralysis; that is, a paralysis of motion on one side of the body and a paralysis of sensation on the opposite side. The disorder of sensation reached as far as the third rib and involved the arm in front and behind, and though the analgesia and anesthesia were not complete, there was complete thermo-anesthesia; in other words, there were hypalgesia, hypesthesia, and thermo-anesthesia.

The examination of his motor side revealed partial right hemiplegia with an exaggeration of all the tendon-jerks, a right ankle clonus and a left ankle clonus, neither of them exhaustible; the right knee-jerk was extremely active, the left very active. There was the big toe phenomenon on the right side. The abdominal reflex was unelicitable. The tendon-jerks of the upper extremities were very much increased. There was no evidence of disorder of function of any of the cranial nerves, nor was there nystagmus nor pupillary abnormality. The optic discs were of normal appearance.

Examination of the heart revealed a loud systolic murmur over the aortic valve, the second sound being sharp and clear. There was a systolic bruit heard at the apex, but this seemed to be the same as at the aorta, since it increased in intensity, over the apex in the right intercostal space. Blood pressure was 120, about the same on both sides. Pulse at that time showed no remarkable abnormality.

In addition to motor and sensory disturbances, there was a distinct and continuous subjective sensation of numbness in the right hand. The electrical reactions of the muscles were practically normal; shoulder and arm all responded to galvanism and faradism in a normal manner.

Laboratory Findings.—The laboratory examinations at the time showed the cerebrospinal fluid to be normal. There were very few lymphocytes found in each smear, an average of less than five in one hundred. Analysis of the blood showed an extremely moderate secondary anemia.

The diagnosis of disseminated sclerosis was made at that time.

The patient left the Institute on August 25th, having

been there five weeks. When he left he was able to walk fairly well. There was no evidence of hemiplegia except that he held his hand in a somewhat awkward position. He was able to stand, dress himself, walk well, and there was no apparent disturbance in gait.

Three weeks after returning home, he suddenly had another attack. In 48 hours the left leg had become as badly affected as the right—stiff, spastic and hyperirritable. There was no further involvement of the upper extremities; the left was normal and the right remained as it had been up to the time of his relapse. This was a distinct attack. He remained in bed three days, after which he began to improve, and improvement was rapid. In about a month, the left leg was practically well again.

In the early part of October, after a warning of 24 hours' duration, during which time he was aware of a peculiar feeling of weakness, he became fully quadriplegic, and for quite a length of time he was unable to dress himself, to feed himself, and so forth. One side was as badly involved as the other. There was no pain, nor has he complained of pain in any of the attacks. His brother states that there was the same extent of hypalgesia that there was before.

On the last of October, he was practically well again. Recovery was gradual but almost complete. He was able to use his hands and arms very well, he sat up with the family and even played cards.

The quadriplegic attack was on October 12th, and it is from this latter attack that he is now recovering. There has been a decided change, however, in his sensory disturbances. The chart plotted at the Neurological Institute shows that at this time he is completely devoid of tactile, thermo and pain sensibility in the areas corresponding to the fourth dorsal segment.

The physical examination, aside from that shown on the chart, is as I have related it.

I will call your attention to the position of the feet. They are in that state which we come to look upon as a static motor irritation. The terminal phalanx of the toes is flexed, there is a position of *pied bot,* as the French writers call it. He has some slight capacity to move his left foot; there is no capacity, however, to lift the right. Tendon-jerks are very much exaggerated on both sides. There is a double and inexhaustible ankle clonus.

The Oppenheim phenomenon is elicitable on the right side. There has been on this side a distinct Babinski, which, however, was not distinct a few minutes ago when I carefully reëxamined the patient. Doctor Baehr examined him a few days ago in his home and the big-toe jerk was very distinct. It may have been that the foot was cold when I attempted to elicit it and this sometimes causes alteration in the Babinski sign. The motor hyperexcitability extends throughout the entire body and is very nearly as marked in the upper extremities as it is in the lower extremities. It does not, however, extend to the jaw. The jaw-jerk is not particularly increased. There is now no astereognosis. He detects objects as well in the right hand as in the left. There is very little, if any, evidence of the former paralysis in his hands at present. Indeed, I think there is none whatsoever.

The ophthalmoscopic examination when he was in the hospital, was, as I have said, negative. He has been examined since then, but there is no nystagmus and no pupillary defect. The only other existing objective sign which I have not pointed out is the absence of his abdominal reflexes.

There has been no involuntary drawing up of the legs in bed. A sensation of trembling or fibrillary feeling ex-

ists in the legs as if a wave were running up and down. There has been but little trouble in holding the contents of the bowels or in making water.

I have never been able to find any disorder of his deep sensibility. The typhoid fever, which he had for three months when he was fifteen, was characterized by great delirium and high temperature, and he has now some scars on his knees, which, he says, resulted from that attack. His cardiac condition, as I have said, is purely a valvular one; there is no disorder of the nerve supply nor of the competency of the heart muscle.

His recovery has always been a gradual one. The exaggeration of the tendon-jerks, the clonus, the Babinski have never disappeared; for instance, his arm has practically recovered but it still has the same evidence of hypertonicity which it has always had.

I have not said anything about any speech disturbance. There is a suspicious slowness about his articulation, but his brother tells me that he has not noticed any abnormality. There is a tremor of his hands, but it is that of a person who has been ill, and is not an intention tremor. There has never been an ocular palsy or persistent diplopia or disturbance of chewing or swallowing, nor have there been any spasmodic attacks of hiccough.

Diagnosis.—One of three diagnoses may be made in this patient:

(1) Disseminated sclerosis.

(2) Hemorrhage into a pre-existing syringal cavity.

(3) Disseminated myelitis.

The more familiar we become with the disease now commonly spoken of as disseminated sclerosis the more we appreciate the manifold forms in which it displays itself. In every instance in which there are symptoms of subacute or chronic myelitis it should be suspected. In

this case the abrupt onset of the disease, attended with mild febrile disturbance, suggested an infection and possibly a disseminated myelitis, but the aftercourse of the disease is opposed to this assumption. One can imagine that a healthy, strong young man may have a syringal cavity in his cord and not display indication of it, and that into that cavity hemorrhage may occur, the process of disintegration extending into the vessels beneath the walls of the cavity; but the repetition of the attacks and the degree of recovery which the patient made were both against this supposition.

On the other hand the onset of the disease is very unlike the onset of the average case of disseminated sclerosis. But the essential thing for us to learn and to admit is that the average case of disseminated sclerosis is not necessarily the prototype of the disease. There are any number of cases on record in which disseminated sclerosis began with clinical manifestations of what are considered to be acute myelitis. In the second place the course of the disease, that is, its characterization by exacerbation and remission, is very characteristic, and in the third place the degree of recovery which is made, that is, the quiescent state into which it has now passed, is very common in disseminated sclerosis.

TUMOR OF THE CAUDA EQUINA IN A SYPHILITIC, SIMULATING SACRO-ILIAC ARTHRITIS

C. BURNS CRAIG, M.D.

The following case illustrates the manner in which the symptoms of a slow growing tumor of the cauda equina develop. The patient, a hotel manager, 49 years old, complained of aching pain in the left side of the small of the back, and in the left lower extremity, which was increased by stooping; of an increasing numb, dead feeling in the left foot and leg; of difficulty in starting the stream of urine; and of failing potency.

The patient was formerly an immoderate user of alcohol, and gives a history of syphilitic infection at the age of 23 years, for which he received two years of indifferent treatment with mercury.

The symptoms of the present disorder began ten years ago, with a slight ache in the small of the back, diagnosticated rheumatism and treated with a battery for several months. The pain was absent or insignificant during the day but would waken him at night. He frequently arose and walked up and down the room, thereby obtaining relief.

About a half year after the onset he was awakened by an attack of excruciating pain low down in the back, so severe that he thought he was going to die. Walking gave no relief. He went to bed and applied hot-water bottles and hot blankets, but the pain continued until the middle

of the following morning, when it mitigated so that he was able to go about his work.

Except for attacks of very slight pain, he was well the following 4 months. He was then stricken with an attack of pain in the same region, of greater intensity, which lasted about 4 months. He was unable to sleep in bed, but lay propped up on a lounge. The pain often continued during the day so that he was compelled to lie down. Up to this time he had had no difficulty in standing or walking, nor any bladder disturbance. The following spring, about March, he was seized by another attack similar in nature. While the pain lasted, walking was almost impossible, but when he was free from pain he could walk any distance without fatigue.

During the succeeding 4 years he suffered from similar attacks of severe pain, usually coming on in the spring-time and lasting from 6 to 10 weeks. He went to numerous physicians and was always treated for rheumatism. About 4 years ago the pain subsided for a period of 18 months. During this time he had pain only when stooping.

One evening in February, 1914, while sitting in a cold room, he began to have a slight return of his old pain, which gradually increased in severity and lasted all night. No relief was obtained in any position, nor by standing nor walking. He was in bed 6 weeks with the most severe attack of pain which he had yet experienced. During this attack the pain was not confined to the back, but would radiate from that spot down the thighs to the knees. The ankles swelled and he was not able to wear his shoes. On two or three occasions he had difficulty in starting the stream of urine, attended by severe pain. He was also obstinately constipated. In April of the same year he went to a sanitarium for rheumatism, during which

time he could not lie down, but tried to sleep in a sitting posture. While there he also developed a sharp pain in the right shin bone, which extended later up the thigh. The leg became greatly swollen and an ulcer appeared on the calf which discharged for about a month. The swelling about the ankles disappeared while the man was in the sanitarium, but the pain persisted. A few weeks later he went to Mt. Clemens for 2 months, where he took baths and osteopathic treatment, without benefit. During December, 1914, and the succeeding 2 months, he continued osteopathic treatment, and improved so that he was again able to walk a mile or more with pain, whereas before he was unable to walk at all. During 1915 he would have an occasional attack of severe pain, but managed to continue his hotel work and was able to sleep lying down. Constipation practically disappeared, but he would occasionally have difficulty in passing water.

In June, 1916, he noticed for the first time a numbness in the toes of the left foot, as though the shoe were too tight. About 6 weeks before he came to the Neurological Institute he noticed that he could not wiggle the toes of the left foot as well as those of the right. For 2 years the left leg had been getting thinner, and he had lost about 35 pounds in weight.

Physical Examination.—On physical examination the pathological signs were limited to the left lower extremity and the cardiovascular system. The latter showed a slightly hypertrophic heart, moderate fibrosis of the arteries and systolic blood pressure of 230 mm. of mercury. The patient, a rather heavily built man, limped as he walked, bearing his weight on the right leg. The left knee was lifted high to avoid catching the dependent toes. There was slight uncertainty of station but no defi-

nite Romberg sign. There was distinct atrophy of the left leg, thigh and gluteal region. The right knee-jerk was slightly greater than the left. The right ankle-jerk was not obtained, the left was present. There was feeble movement of the extensor longus digitorum, but none of the tibialis anticus on attempting to extend the foot. The pupils were slightly irregular but responded promptly to light and in accommodation.

The sensory examination showed a mild degree of hyperesthesia over the left lower extremity as far up as the middle of the thigh anteriorly, and extending up over the left buttock posteriorly, and including also the opposite buttock. This hyperesthesia was present to all modalities of stimulation. There seemed to be no disturbance of sensation over the scrotum, but the penis was hyperesthetic.

Electrical examination showed slight general reduction to faradism, slightly more marked in the muscles of the left lower extremities below the knee. The contractions in these muscles were less forcible and less prompt, particularly the peroneal and anterior tibial group. The galvanic response was prompt and active. There was no reaction of degeneration.

Laboratory Findings.—The serological findings were: serum Wassermann negative, spinal fluid Wassermann 2 plus, globulin in excess, cells 2, Fehling's reduction plus. The urine, specific gravity 1.018, no albumin, no sugar and no pathological microscopic findings.

In spite of the plus Wassermann in the spinal fluid and the history of syphilitic infection 26 years ago, the case was diagnosticated as tumor of the left roots of the cauda equina. On December 14th, 1916, laminectomy was performed by Dr. Charles A. Elsberg. When the dura at the level of the tenth dorsal vertebra was exposed it

was seen to be tense and bulging, and a dull, bluish colored body was visible beneath it. Incision of the dura exposed a moderately soft, bluish tumor mass, 1 x 2 x 3 cm. lying upon the conus and roots of the cord, which was fairly easily removable and came out in toto.

Subsequent History.—Convalescence from operation was uneventful and satisfactory. Following the operation the patient experienced considerable pain in the left lower extremity and both extremities were for a few days completely paralyzed, as was also the bladder. In the course of the next month the right extremity and bladder recovered completely, and recovery in the left extremity began. At the present time, July 1, 1917, the patient can stand and walk securely. He still has a moderate degree of pain in the left lower extremity at night, and slight difficulty in urinating.

In addition to being of interest to show how long these cases exist without recognition, this case illustrates how necessary it is, now that our perception of syphilis of the nervous system is so keen, not to charge everything that befalls a syphilitic individual to syphilis.

DISSEMINATED SCLEROSIS. CHRONIC MYELITIS OR TUMOR OF THE SPINAL CORD

J. L. JOUGHIN, M.D.

The patient, an unmarried female 44 years old, of Irish extraction, is employed as a children's nurse. So far as she can recall she was never ill until the first symptoms of the present malady manifested themselves.

Chronologically, her symptoms may be thus enumerated:

First—Pain confined to the outer portion of the left ankle, beginning seven years ago. This was not severe. At first intermittent, it later became more constant and extended to the left knee. It was considered by the patient to be rheumatic.

Second—Three years ago a pain exhibiting similar characteristics developed in the right knee, but never became so marked as the pain in the left knee. These pains have persisted, but have not markedly increased in severity and have been limited to these articulations. They do not occur when she is lying down, but are induced by standing and especially by walking. Changes in the weather neither intensify nor alleviate them, and there has never been any local tenderness or swelling of the parts.

Third—During the last year, to these subjective sensory symptoms have been added a very definite series of subjective and objective motor symptoms, and the condition of the patient has changed markedly for the worse.

These new symptoms were initiated twelve months ago by a peculiar feeling of movement, a tremor or jerk felt in the right ankle, the right calf, the anterior tibial and peroneal groups of muscles. She thinks she has seen actual movement of these parts, but she is not sure of it. Soon similar sensations were complained of in the left leg, but these occurred less often and were less severe. Simultaneously with the development of these subjective symptoms, the patient began to trip over carpets and other objects. Her legs felt stiff and weak and she experienced much difficulty in going up or down stairs, owing to inability to flex the legs at the knee. She walked with a decided limp, and the distance she could cover without a giving way of the legs rapidly diminished. When lying down, especially after retiring, the legs would suddenly involuntarily flex or extend. No day went by in which this did not occur on several occasions. These phenomena were progressive and were present in both legs, but much more in the right, and it is the functional incapacity of this leg which makes locomotion so difficult.

Fourth—An imperative necessity for immediate micturition had manifested itself within the last eight or nine months, but she had never had any involuntary evacuation of the bowels nor bladder, nor had she at any time urinated before reaching the closet. Closely questioned, she insisted that this impairment of function was no worse now than it was six months ago.

Fifth—The last symptom, one dating back to the summer months just passed, consisted of tenderness of the calf muscles which annoyed her each time she dried her limbs after bathing. This existed when she entered the hospital, but has largely disappeared at this time.

Physical Examination, November 5, 1916.—The cranial nerves were intact. The left pupil was a trifle irregular,

but otherwise the eyes were quite normal, including the fundus findings. Speech was unaffected. The upper extremities were in every way normal. There was no Romberg. In the lower extremities there was little if any loss of muscular power, and when the patient was lying down the legs could be elevated to an angle of 90 degrees. There was no incoördination, no tremor, no atrophy, no fibrillation. There was a definite involuntary spastic condition of both legs. The knee-jerks were lively, the right greater than the left, but the left ankle-jerk was greater than the right. On the right, the Babinski big toe phenomenon was elicitable, but on the left this was doubtful although on previous examinations it had definitely existed. The Oppenheim and Schäfer method on producing extension of the great toe were effective on the right. There was a true ankle clonus on the left, and on the right a pseudoclonus. (A few days later a true clonus on each side could easily be demonstrated.) The abdominal cutaneous reflexes could not be elicited. The gait was typically spastic, the right lower extremity displaying it more than the left. The right leg indeed was barely flexed in walking. The toes and anterior portions of the feet scraped along the ground in consequence of the marked spasticity. Neither station nor gait was suggestive of cerebellar disease. The patient presented a marked double planus. Sensibility was intact.

Diagnosis.—The lack of anything in the history to suggest syphilis, the absence of symptoms which are customarily produced by syphilis of the nervous system, and the negative serology exclude lues.

Disseminated sclerosis of paraplegic display it is impossible to exclude. It is true we have no intention tremor, no nystagmus, no speech disturbance, no remissions, no pallor of the optic discs, but these diagnostic

signs of multiple sclerosis are often lacking in cases which later on become quite typical in their clinical display. After seven years we might fairly expect some of the above symptoms to be present, but the course of multiple sclerosis as we know varies within exceedingly wide limits. There is also the probability that this illness is not of seven years' but of one year's duration, that is to say, only since the onset of the objective motor phenomena. We cannot be quite sure that the pains that she complained of for six years previous to the development of the motor syndrome, considering their character and the fact that they occur only when the patient stands or walks, are not dependent upon the double pes planus. In fact this is a reasonable explanation for them. The slight urinary disturbance does not invalidate the diagnosis of the multiple sclerosis, as it is of a grade not uncommonly met with in that disease.

Have we a slow compression of the cord due to neoplasm or to Pott's disease? The latter may be easily excluded. The patient is in excellent health and is gaining in weight, her hygienic surroundings are all that can be desired, the spinal column is neither rigid, tender, nor deformed, the family history is free from any taint of tubercular trouble and roentgenographs of the spine show no pathological alteration.

We may be dealing with an intra- or extramedullary neoplasm in spite of the absence of "root pains" or objective sensory disturbances indicating a new growth. We know how widely variant from the classical picture the sensory manifestations of neoplasm may be, and how in exceptional cases they may never occur. Of late we have begun to recognize that the presence of pain is not always essential in making this diagnosis and that any progressive spastic motor paralysis, especially if accom-

panied by even slight objective sensory disturbances, must always be regarded potentially as due to neoplasm and its future development scrutinized closely if we do not wish mistakenly to denominate all such cases as myelitis, multiple sclerosis, and so forth. In connection with the possibility of new formation it is interesting to note that four years ago uterine "fibroids" were removed from the patient, *i.e.*, three years after the development of the (postural?) pain in the legs and three years before the development of the first symptoms of spasticity. Cases are not unknown which have been repeatedly diagnosed during life as multiple sclerosis and which the autopsy findings have shown to be cord tumors. However, such cases are exceptional, and while we should not forget the possibility of their occurrence, in such cases as this the best diagnosis is disseminated sclerosis. Were there definite sensory disturbances pointing to lesion of a certain segment of the cord, or should these develop later, one would be justified in doing an exploratory laminectomy in order to exclude cord tumor.

PARAMYOCLONUS MULTIPLEX

SANTE NACCARATI, M.D.

Paramyoclonus multiplex, a disorder or disease first described by Friedrich in 1881, is a condition or disease that is of very rare occurrence in this country. Very little is known of its nature or of its dependency. Like all other nerve diseases, in some instances, there seems to be a more or less definite relationship occasionally to fright, trauma, and infectious disease. A familiar variety of myoclonia has been described by a number of German and Italian writers, which is associated with, or terminates in dementia. It is not unlikely that all the myoclonic disorders are an expression of disturbance of the internal secretions, but our knowledge of the internal secretions is not yet so specific that we can say which one is primarily at fault, nor have we advanced so far in experimental therapeutics as to be able to say with any positiveness that administration of one or other of the glands that furnish the internal secretions would be beneficial in any case.

The patient, an Italian twenty-five years old, was employed until eight weeks ago as kitchen-boy in a hotel. He has been married five years, and his wife has had one child, no miscarriages.

So far as can be ascertained, there are no cases of nervous diseases in his antecedents or in his brothers and sisters. He denies luetic infection and his blood Wassermann is negative. He comes from the part of Italy

where cases of Dubini's disease are frequently encountered; but he never had malaria nor any toxic, infectious, or dyscrasic condition while he lived there. In 1916 he suffered from inflammation of the throat, presumably diphtheria.

The symptoms of which he complains now began nine weeks ago when he suddenly noticed slight contractions in the right hand, forearm and arm, which he had never felt before, and which interfered with his work. Similar muscular twitchings soon appeared in the left upper extremity and in the lower extremities, so that his station and gait were somewhat affected and he had to give up his occupation. In addition to the movements of the muscles he noticed an increase of perspiration, sensation of heat in both hands and feet, especially during the night, and a severe pain in the lumbar region, which had been characterized and treated as rheumatic.

Physical Examination.—Physical examination shows tachycardia, a coarse, rapid manual tremor and slight inequality of the pupils, which, however, react promptly to light and accommodation. The tendon-jerks and superficial reflexes are rather lively; there is no Babinski, no Hoffman. General strength does not seem to be impaired.

The chief feature of the case was the rapid violent contractions of all the muscles of the upper and lower extremities and trunk, as though irritated by an electric current. The right side was affected more than the left. The muscles of the face also participated. The contractions were clonic, brief and lightninglike, asymmetrical, unequal, arrhythmical, paroxysmal. In some muscles more than one hundred contractions per minute have been counted. They are subject to some degree of control, and they diminish during physical exercise.

His mental condition seems somewhat deteriorated. His memory is poor, ideation and perception slow, attention indolent. He answers hesitatingly questions in his own language, and many questions must be repeated in order to be understood. The patient, however, relates that his memory has always been poor, so that it was impossible to know whether we were dealing with an inferior intellectual type or whether the mental deterioration was a part of the whole symptomatology.

The electrical reaction of the muscles was normal. There was not the slightest suggestion of reaction of degeneration.

Diagnosis.—As has been said, the pathology of the paramyoclonus multiplex is still unknown. The anatomical findings of some authors, such as Murri—who found a chronic localized pachymeningitis with atrophy of the Rolandic cortex in one case—and of others, are contradictory. All that we know is that it probably has some connection with disorder of the parathyroid. Biedl, in his book on the internal secretions, classifies myasthenia gravis and myasthenia periodica under dysfunctions of the parathyroids, and among the hypofunctions, paralysis agitans, tetany, myoclonus, epilepsy and myotonia.

The direct dependence of tetany upon absence or hypofunction of the parathyroids was demonstrated by the physiological experiments of Vassale and Generali in 1896, by the researches of Cristiani, and by the transplantation of parathyroids done by Eiselberg, Halsted, C. H. Mayo, and others. Out of nine human transplantations three cases of tetany were successfully cured, and in three others the symptoms were remarkably lessened. We have no proofs to confirm the dependence of myoclonus upon hypofunction of the parathyroids, but

we are justified in attempting human transplantations to confirm or reject the Biedl hypothesis. Up to the present time I have been treating this patient with large doses of quinin and with parathyroid gland extract, but without result.

SCLERODERMA OCCURRING IN AN INDIVIDUAL WITH EXOPHTHALMIC GOITRE

E. WHATELY APPLEBE, M.D.

It has been assumed that the strange disease known as scleroderma is dependent upon disordered internal secretion, but the only substantial support of such an assumption is the fact that certain cases have seemed to be improved by the administration of an extract of the thyroid gland. The association of demonstrable disease of the thyroid gland and scleroderma is apparently uncommon. Very few cases are to be found in the literature, and the case herewith reported is the only one that has been met with in the Neurological Institute. It is interesting to note that the manifestations of scleroderma first developed after the enlarged thyroid gland began to diminish in size.

The patient, an Italian woman 49 years old, the mother of four healthy children, was well and strong until she was thirty-four years old, when she developed an enlargement of the thyroid gland, which progressed up to two years ago, when it began to get slightly smaller.

Two years ago, while passing through the menopause, she had a severe attack of rheumatism involving a large number of joints, especially those of the upper extremities. As this attack cleared up and the swelling was going out of the joints, she noticed that the skin over the fingers was growing smoother and tense, and that she was unable to flex the fingers. It was for this unwieldiness of the

fingers and hands, and tightness of the skin over the forearms, and a sense of general immobility, that she came to the clinic.

Physical Examination.—The patient, a large, fairly well nourished woman, displayed a curious mask-like expression of the face. The eyes were deeply sunken, the skin being tightly drawn over the orbital margins, and the tissues around the mouth and over the cheek appeared to be partly atrophied. At the margin of the lips there was a definite groove at the line of demarcation between the lip and the surrounding skin. Her face in repose was expressionless, but the emotions could still be well expressed. The goitre was about the size of a small lemon, the right lobe being chiefly affected.

On palpation over nearly the entire body and extremities, the fingers were met by a distinct sense of resistance, and the surface temperature of the body appeared below normal. The fingers presented a typical picture of sclerodactylia. The skin over them was smooth and glistening and appeared to be tightly bound down to the underlying bone. The atrophy of the subcutaneous tissues made the joints appear enlarged. The phalanges were in a slightly flexed position, and could not be further flexed or extended. The nails were not involved.

The skin could only be picked up in folds over the parts of the body least affected. Over the hands it was impossible to raise the skin from the subjacent structures at all. The mucosa of the mouth and the vagina was unaffected, and there was no kraurosis vulvæ.

The deep reflexes were normal, the cutaneous rather sluggish. There was a fine tremor of the fingers, hands and tongue, and an entire absence of any sensory disturbance.

Laboratory Findings.—The pulse rate was 100 to 120,

and the blood pressure 200 mm. The heart showed marked arrhythmia. The blood count showed a white cell count of 10,800. Differential count, neutrophile 60 per cent; eosinophiles, 2 per cent; large lymphocytes, 20 per cent; small lymphocytes, 16 per cent; transitionals, 2 per cent. Urine: specific gravity 1.030; slightly acid; trace of albumin.

In addition, the woman had a brownish discoloration extending from the middle of her legs down over her feet, becoming darker around the ankles. She has had varicose veins in her legs for many years, and this discoloration is doubtless due to that condition and is merely an accidental accompaniment of her other affections. There were no mental symptoms.

Diagnosis.—The interesting point in this case is the relationship between the scleroderma and the goitre. The patient comes from Milan, and in that section of Italy goitre, while far from being as prevalent as in Austria-Hungary and certain parts of Switzerland, is still frequent enough to be regarded as endemic.

Scleroderma, like goitre, occurs about nine times as frequently in women as in men. In an autopsy on a case of scleroderma performed by Dr. Singer, degeneration of the thyroid was found, and Dr. Beer reports a case associated with exophthalmos and rapid heart action.

The pathology of this condition consists of a condensation of connective tissue elements. The deeper layers of the derma are also affected. In well marked cases the elastic fibers are increased. The fat cells and glandular structures finally become atrophied. There is a diminution of the caliber of the vessels due to fibrosis, and in the later stages the nerves also show a fibrous proliferation.

In conclusion it may be said that as our knowledge of

the endocrins increases and cases are more carefully studied and followed up, it appears more than probable that a definite relationship between scleroderma and dysfunction of the thyroid will be established.

THE DIAGNOSIS OF BRAIN TUMOR

JOSEPH COLLINS, M.D.

The diagnosis of brain tumor is often an easy matter. On the other hand to say, with confidence, just where it is situated in the brain is always difficult. Occasionally we encounter a patient whose preponderant symptoms are tumor, but who has also symptoms which suggest other disease, and especially vascular disease of the brain. This association of symptoms occurs now and then in cases of brain tumor after rupture of a blood vessel of considerable size has taken place within the new growth. The symptoms that are the result of the vascular lesion then obscure the otherwise distinct clinical picture. In other instances the difficulties of differential diagnosis are increased by the simultaneous existence of unrelated disease and particularly disease of the kidneys. We recently had an experience which illustrates this difficulty of differential diagnosis.

A married woman, 43 years old, was admitted to the Neurological Institute, November 1, 1916, complaining of headache, lethargy, stuporousness, attacks of vomiting, and inability to move the left half of the body. These symptoms, which had been in existence for upward of two months, had been gradually progressive.

She had been a hard-working dressmaker, who had experienced much stress and disappointment in life. She had not had infectious disease, nor had she encountered physical injury. She considered herself in good health

until the beginning of the present illness, in the latter part of August, 1916, when she began to complain of weakness of the left upper extremity. It was particularly noticeable in the hand when she attempted to work, but it was not so incapacitating as to cause her to give up her work or to lead her to consult a physician. After it had been in existence a week or more, she awakened one night to find her left upper fingers in a state of mild clonic convulsion, which extended throughout the entire extremity and displayed itself also in the left side of the face. The attack lasted a few moments, and there was no disturbance of consciousness. She went to sleep after it was over and the next morning did not experience any ill effects from it. It occurred again the next night, and the following morning the left upper extremity felt decidedly weak. She then called a physician, who, finding a trace of albumin and some hyaline casts in the urine, diagnosed the condition as one of uremia, and gave her appropriate treatment. The weakness of the left upper extremity continued to increase, and a few days after the attack of Jacksonian epilepsy she complained that the left leg felt heavy and that she was obliged to drag it in walking. She then began to have headache, of gradually increasing severity, and at the end of a week she began to have nausea and attacks of vomiting. The headache and vomiting continued almost incessantly for three weeks; then the latter ceased, the former mitigated very considerably, and she became very apathetic. When she refrained from eating she vomited rarely. On account of the vomiting and voluntary abstinence from food she lost flesh and strength rapidly.

When she was admitted to the hospital on November 1st, she was quite apathetic, answered questions responsively and with apparent accuracy. She seemed to tire

very easily on attempts at conversation, and a phenomenon was then first noted which afterward became rather dramatic: during the process of answering her response would suddenly become slow, her eyes would semiclose and she would go rapidly into what seemed to be a state of overwhelming sleep. She would arouse when spoken to sharply or when shaken, and then for a few minutes the same obscuration of consciousness would be displayed. When not in this state she was always completely oriented, able to recognize people by the voice tones or character of the step, as well as by looking at them. She related that the headache had not been very depressing for several days past, and that she did not vomit when she refrained from putting food into her stomach. She did not complain of dizziness.

Physical Examination.—Physical examination revealed a left-sided hemiplegia of slightly spastic character; the hemiplegia was quite complete. She was unable to stand and apparently could make no voluntary movement of the left arm or leg. The tendon-jerks were livelier on the left side than on the right, but were not exaggerated. The Babinski big toe phenomenon was elicitable on the left side, but not on the right. The abdominal and epigastric reflexes were not elicitable. There was no digital reflex either on the right or on the left side.

It was necessary to embrace the opportunity when she was mentally alert to make the sensory examination, but it was satisfactorily established that there was no disturbance of cutaneous or deep sensibility, although there was distinct astereognosis of the left hand.

There was no disorder of the senses of smell, taste or vision, but the ophthalmoscope revealed a double choked disc of about two diopters' elevation with hemorrhages limited to the discs.

The pulse was 56, the blood pressure 110, the heart sounds clear, the arteries not thickened. The patient was incontinent, so that it was impossible to estimate the amount of urine secreted, but it contained a trace of albumin and a few hyaline casts. The Wassermann examination of the blood serum was negative, and the cerebrospinal fluid which did not escape through the needle under pressure was quite normal.

While the lumbar puncture was being made she had a convulsion consisting of slight clonic movements in the left side of the face, the head and eyes turning to the left. She became unconscious for about two minutes and then vomited. When she regained consciousness she had no recollection of the attack. The next day it was noted that the stupor was becoming deeper, and that she exhibited definite automatic movements of the right upper extremity while in the stupor. She was somewhat arousable, and on questioning her when aroused she answered at first responsively, but after a minute or two her replies became very slow, after which she exhibited the automatic movements just spoken of as if she were brushing something away. She complained of severe pain in the right side of the head, and her family now corroborate her statement that the headache has always been most severe on that side.

The diagnosis of brain tumor was made and consent was obtained for exploratory operation. After she had been prepared for operation the patient's general condition seemed so unfavorable and the evidences of the delimitation of the alleged new growth were so inconclusive that it was decided to postpone it. About a week after she was in the hospital it was noted, after a second lumbar puncture, that she seemed considerably brighter. She did not complain so much of headache and she rarely

vomited. The mental hebetude and incontinence of urine and feces constituted her chief symptoms. Although there were no somatic evidences of syphilis whatsoever and although the blood serum and cerebrospinal fluid were quite negative it was decided, because of certain features of her history, to give her a provocative dose of salvarsan, which was administered on November 1st. The results of that administration did not further the suspicion that we might be dealing with a syphilitic process.

On the 23rd of November it was noted that she had seemed more alert for the past few days, and that again this alertness had followed the withdrawal of 20 c.c. of cerebrospinal fluid which came out on this occasion under very considerable pressure. It was likewise noted that the ophthalmoscope now showed a number of hemorrhages into the discs. The papilledema remained about two diopters. The macula was free from any change.

Examination a week later showed a very striking alteration of some of the physical signs. Whereas up until now the hemiplegia had the features of moderate spasticity, at this time they were those of flaccidity. When the patient sat up in bed her head flopped back as would that of a hydrocephalic child. The entire left side was flail-like. Neither knee-jerk could be elicited. The left ankle-jerk was absent, the right present though sluggish. Irritation of the left sole caused display of a typical Babinski phenomenon of the right sole flexion. The left triceps jerks could not be elicited, nor was there any other arm response to the blow of the percussion hammer over the supinators. The tendon-jerks of the right upper extremity were elicitable. The abdominal and epigastric reflexes could not be elicited. Examination of the sensory sphere was carefully made at this time, and no

disorder could be made out. The patient recognized the contact of objects in the left hand, but she could not distinguish them, even when her fingers were pressed around such objects.

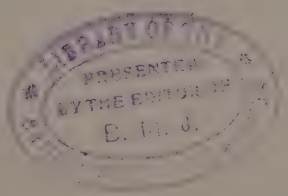
A very noteworthy feature of her condition was that shortly after her entrance into the hospital, namely, on the 3rd of November, her blood pressure began to go up from 110 so that on the 6th it was 150. It remained thereabouts until the 9th, when it dropped down to 120 and fluctuated between that and 100. Her pulse rate varied from 50 to 60, say about 56.

After the first of December she gradually became very stuporous, no new symptoms developed, and she died on December 12th in a state of complete exhaustion.

Diagnosis.—Although there was little hesitancy in making the diagnosis of brain tumor and allotting its location to the middle of the right precentral convolution, nevertheless the existence of a definite nephritis had to be given full consideration. The important symptom that was wholly irreconcilable with the assumption that the symptoms were the cerebral manifestations of uremia, was the Jacksonian epilepsy. This was typical in its display that it could only be the manifestation of an irritation such as might be produced by an organic lesion irritating the lower third of the precentral area. It was not so easy, however, to exclude focal softening in this area. We had seen practically the same symptoms, except the extraordinary fading away of consciousness while actively cerebrating, and the optic neuritis, in a woman of the same age. Autopsy showed them to be dependent upon softening of the Rolandic area. In the case under consideration the fact that nephritis existed had to be given full weight. Occasionally papilledema and hemorrhage in the disc occur in nephritis, and assuming that

the nephritis caused the ocular manifestations in this case, it only remained to explain the vacillation of consciousness in order to reconcile the symptoms with focal softening.

The absence of any demonstrable vascular range, the existence of bradycardia, the insidious onset of the symptoms, and the retrogression of the spastic phenomena successfully militated against the assumption that the symptoms were due to focal softening. Despite the fact that the seeming acute onset of the disease suggested the dependency of the symptoms upon vascular lesion, when attempt was made to estimate the nature of the neoplasm and the degree that it had encroached upon the brain, the important facts were that the motor symptoms, starting with slight enfeeblement of the left hand, had progressed within a fortnight to complete hemiplegia. One of two things must account for it: either there was a rapidly growing lesion causing infiltration of the motor cortex, or there was a small growth into which a hemorrhage had occurred. When the spastic phenomena were replaced by the flaccid, and the stuporousness rapidly increased, the latter was assumed to be the explanation.



THE DIAGNOSIS OF DISSEMINATED SCLEROSIS

JOSEPH COLLINS, M.D.

The diagnosis of disseminated sclerosis is made from consideration of all the symptoms, the way in which they have developed and comported themselves after development; from careful analysis of the objective symptoms or physical signs; and finally by process of exclusion, that is, by eliminating the possibility of other diseases of the central nervous system which may give rise to symptoms similar to those which the patient presents. When we consider that the islets of connected tissue constituting the lesion of the disease may be situated in any part of the central nervous system, and that they are invariably multiple, it is not astonishing that disseminated sclerosis may parallel in its clinical display any organic or functional disease of the nervous system. For instance, such islets situated in the posterior column of the spinal cord may give rise to the subjective and objective symptoms of tabes; situated in the lateral tracts of the cord and simultaneously in the ventral gray matter they may cause the phenomena of amyotrophic lateral sclerosis; situated in the ventral portion of the oblongata, they may cause the symptoms and signs of bulbar palsy; situated in the cerebellum they may cause symptoms which suggest cerebellar tumor or cerebellar atrophy, and so on throughout the entire list. Disseminated sclerosis must be diagnosticated by evaluation of the symptoms that are present and by consideration of

the symptoms that are absent. For instance, to distinguish it from tabes we take into consideration the information furnished by study of the blood serum and cerebrospinal fluid, the condition of the pupils, and the sensory disturbance. The cerebrospinal fluid is normal in disseminated sclerosis; it is practically never normal in tabes. The Argyll-Robertson pupil never occurs in disseminated sclerosis; it occurs in eight cases out of ten in tabes. The sensory disturbances in tabes have a rather characteristic display corresponding with the distribution of the nerves that enter the segments of the spinal cord affected by the process. Thus, we reach a correct diagnosis in any given instance by eliciting and weighing the evidence. The procedure is very analogous to that of the law. The facts alleged or actual are elicited, their probability or credibility is discussed and debated and their number estimated, when the decision is rendered in conformity with the evidence, its weight and credibility. Oftentimes the verdict must be rendered on circumstantial evidence, and though circumstantial evidence is not so important in medicine as in the law, it is nevertheless of great value in leading to correct diagnosis.

The object of diagnosticating disease is first to be in a position to adopt measures that will mitigate its course or eradicate it, and second that we may be in a position to answer the patient's question, "What ails me, and will I get well?" Although the facts, that disseminated sclerosis is a disease whose attribution or origin is entirely unknown and that its course is wholly uninfluencable, thwart the first object, it must, however, be borne in mind that in making the diagnosis we eliminate the existence of a disease of similar display which is founded

upon morbid process susceptible to cure. Hence the incentive to correct the diagnosis is just as great.

The islets of sclerosis constituting the anatomical basis of the disease may be situated in any portion of the central nervous system. It was formerly thought that they had a predilection for certain locations such as the vicinity of the ventricles, the corpus callosum, and the posterior columns of the cord; but there is little real foundation for such a statement. As a matter of fact, we do not know anything of the factors that cause deposition of the sclerotic foci, or whether their development stands in any relationship to the blood vessel supply of the parts or not. If disseminated sclerosis has any distinctive characteristic, it is multiplicity of symptoms, which of course is the natural result of multiplicity of disease foci. Charcot familiarized the profession with the fact that oftentimes the disease displayed itself by spasticity of the extremities, scanning speech, intention tremor and nystagmus. Nothing hindered the recognition of the disease so much as the widespread belief that these symptoms must exist before the diagnosis could be made. I have not infrequently said that if we did not make the diagnosis without eliciting such symptoms, we would miss at least 75 per cent of the cases. The safe rule to follow is to suspect disseminated sclerosis in every instance in which spasticity of the extremities develops without clear attribution. If in such instances it can be elicited that the patient has had, even remotely, such symptoms as transient diplopia, indefinite and transitory paresthesia, and fleeting disorder of equilibrium, the probability that we are dealing with the disease, disseminated sclerosis, is very great. This probability is enormously enhanced if the individual is found on examination to have any one or more of the so-called classi-

cal symptoms emphasized by Charcot, and particularly if on examination there is found to be an absence of the abdominal reflexes.

Disseminated sclerosis, it matters not in what guise it displays itself, has certain features that are highly characteristic, and familiarity with these features facilitates recognition of the disease, or assures us of its reality when suspected. The first and most distinctive feature is the insidious way in which it begins. Perhaps in as many as eight cases out of ten it is very difficult for the individual to fix the time when the symptoms, which he afterward realized were merely indications of the beginning of the disease, first displayed themselves.

The next most characteristic attribute of disseminated sclerosis is the course of the disease. It rarely, if ever, progresses uniformly. Its course is characterized particularly by remissions, not necessarily complete cessation of the symptoms of the infirmity, but diminution of intensity of them. In some instances they cease entirely and the individual considers himself well, but invariably they occur and with greater severity than before. It is generally stated that disseminated sclerosis is an afebrile disease, and in the main such a statement is correct. In some instances, however, and this point cannot be too strongly emphasized, the disease has its onset with fever. One of the best illustrations is one of the cases here detailed. It is quite possible that there is a pathological process consisting of disseminated myelitis and encephalitis due to an unknown infection that cannot be distinguished clinically from disseminated sclerosis, but at the present time it must be admitted that 2 or 3 per cent of the cases are attended with fever in their incipiency. It is axiomatic, of course, that fever may occur in dissemi-

nated sclerosis as a symptom of some disorder secondary to the disease, such as from infection of the bladder.

When we come to the question of the differential diagnosis of disseminated sclerosis, we are at once confronted with the fact that it must be distinguished from practically every non-inflammatory disease of the central nervous system and from hysteria. To make the differential diagnosis one must be familiar with the display of such diseases and especially with what is called a typical display. A number of instances are herewith cited as illustrations of how such differential diagnosis has to be made.

DISSEMINATED SCLEROSIS OF THE MEDULLA OBLONGATA

JOSEPH COLLINS, M.D.

Disseminated sclerosis may parallel in its clinical display nearly every disease of the central nervous system. One of the guises that it least often assumes is that of progressive bulbar paralysis. In order that disseminated sclerosis shall produce the clinical syndrome of glossolabiolaryngeal paralysis and no other symptoms, it is necessary that the lesion shall be limited to the confines of the medulla oblongata, and as one of the most constant characteristics of the lesion is its dissemination over a wide area, it is not surprising that we rarely encounter examples of progressive bulbar paralysis dependent upon disseminated sclerosis. We have had such a case under observation for the past several months.

The patient, a Lithuanian carpenter, 38 years old, first remarked when he was 24 years old that at times his legs felt weak and tired without adequate cause, and that at times his arms and hands were the seat of a peculiar numb, dead sensation. The latter was forced upon his attention by his occasional inability to drive a nail properly. He would hit the nail on the head two or three times, and then hit it only partially on the head, or miss it entirely. In other words, manual incoördination was an early symptom. He did not give much heed to these infirmities, and indeed it was not until the more incapacitating symptoms of his disease developed that they were

linked up with it. The second symptom to develop was occasional disorder of gait. At times he staggered as if he were drunk. This was not continuous. It would occur occasionally and last for a short time and then disappear as unattributably as it came. The beginnings of the disease were so insidious and so incontinuous that it was impossible for him to say exactly when the disorder began. He came to this country when he was 32 years old and worked at his trade until he was thirty-five. During this time he had frequently noted a tremor of the right hand which was particularly manifest when he attempted to write his name, but also in doing his work. He also frequently remarked a tendency to stagger. When he was thirty-five he fell off the scaffold and broke his nose and right arm. When he was thirty-six the bulbar symptoms began, the first of them being dysarthria. At times he talked as if he were drunk. Then this would entirely disappear. The next symptom was difficulty of swallowing. Occasionally fluid would regurgitate through the nose; at other times he would have violent coughing spells. About this time, or before, he began to complain that he could not hear so well in the right ear, and of attacks of diplopia. About the same time, possibly before, he remarked that he would have crying spells, even though he did not feel depressed. There has been no complaint of sensory disturbance, pain or anesthesia.

Physical Examination.—He was a hardy, vigorous-looking man, whose facial expression was one of apathy and sorrow. On analysis it was seen to be due to obliteration of the lines of the face. They were ironed out, and the corners of the mouth drooped. On his attempting to talk, the facial musculature did not seem to participate in the action. In other words, the lips, face and forehead

were quite immobile. The lips were semi-opened, and occasionally a little saliva drooled from them. When he attempted to move the lips, the result was a vigorous effort, but little action. He was able to thrust out the tongue vigorously and to move it dexterously, both outside and inside of the mouth. When he attempted to whistle, the lips puckered, but he could not emit any tone. His voice was likewise toneless. The articulation was almost syllabic. For instance, in attempts to say the word New York, he said cwo n (with a nasal tone) rk (with a nasal tone). After he swallowed twice in succession, he had to make violent efforts to swallow the third time. When he attempted to wink either eye, the whole face, including the lower jaw, participated in the action.

On standing with feet together he swayed through a large arc from side to side and backward. It was not the phenomenon of titubation, but of pitching. The gait was markedly ataxic. He walked with feet wide apart and with a tendency to pitch and reel toward the right. There was no definite paralysis aside from the paralysis of the face. There was slight distinct intention tremor of both hands, the right somewhat more than the left. There was no evidence of myokymia. The knee-jerks were active on both sides, as were also the ankle-jerks, and there was a Babinski big toe phenomenon on both sides. The Hoffman finger-jerk could not be elicited.

The right abdominal reflex was present, the left absent. The same was true of the epigastrics. There was slight distinct atrophy of the muscles of the left shoulder girdle, particularly shown in flabbiness of the triceps and biceps. The deltoid was less implicated. The strength of the left upper extremity was diminished proportionately to the atrophy. Alternating movements of the left upper extremity such as pronation and supina-

tion of the hand were badly performed. There was no fibrillary twitching in any of the muscles or any alteration of electric excitability in nerve or muscle. The direct, so-called myotatic, irritability displayed by movement of the muscles when directly percussed was increased, especially in the muscles of the left upper extremity.

Tactile, thermal and pain sensibility were intact. Postural sensibility was considerably impaired, especially on the right side. There was no astereognosis.

The laryngoscope showed that both vocal cords moved very sluggishly on attempts at phonation. The right showed distinct adductor paralysis. The nose and nasopharynx contained considerable quantities of mucopus which ran into the larynx. None of the sinuses transillumined well. External examination of the eyes showed no abnormality save nystagmus. On looking to the right the eyeballs oscillated slowly and rhythmically. When looking toward the left the rhythmic movements were of smaller amplitude. The patient's diplopia seemed to be due to the nystagmus rather than to paresis of any muscles. Examination of the fundi revealed dirty, gray discs, and a hazy retina.

The cerebrospinal fluid was quite normal, and it and the blood serum gave a negative Wassermann reaction.

Diagnosis.—Clinically, the patient presented what is commonly known as bulbar palsy. The dysarthria, dysphagia, aphonia, choking attacks, toneless cough, the immobile face, and the expressionless mouth, all justify that designation. Such a symptom complex may be dependent upon a variety of pathological conditions. The most common one is progressive degeneration of the ganglion cells in the ventral portion of the oblongata. Such lesion constitutes the anatomical basis of the disease

known as Duchenne's disease, or glossolabiolaryngeal paralysis. That disease is invariably accompanied by atrophy of the tongue with fibrillation. In this case there is neither atrophy nor fibrillation. A symptom complex such as this man has may accompany cavity formation in the oblongata,—syringobulbia. In these cases there is invariably characteristic dissociation of sensibility,—thermo-anesthesia and analgesia with relative preservation of tactile sensibility. In this case there is no such disorder. Moreover, in syringobulbia, spastic phenomena, especially of the upper extremities, frequently exist. When they do not exist it is because the muscular atrophy is so profound that spasticity cannot be displayed.

A symptom complex similar to this may likewise exist with vascular disease of the oblongata, and especially when such disease is of syphilitic origin. I have recorded examples in which the chief symptoms of bulbar paralysis were due to basilar meningitis of syphilitic origin. There is no indication of vascular disease in this patient, and the Wassermann reactions of the serum and cerebrospinal fluid are negative. Moreover, since he has no somatic evidences of syphilis, such diseases may be excluded.

Bulbar paralysis is so frequently dependent upon the lesions of amyotrophic lateral sclerosis that it is necessary for us carefully to exclude it. The features that are opposed to such diagnosis are: (1) The duration of the disease. Amyotrophic lateral sclerosis runs its course in the vast majority of instances within five years. (2) Its mode of development. The latter disease usually develops brusquely and pursues an uninterrupted course. In this case it was insidious and irregularly progressive. The facts that the disease has reached an advanced stage without the occurrence of atrophy of the tongue and lips,

and that there is absence of fibrillation in the muscles of the upper extremities, and of lingual tremor, are opposed to it. Finally (3) the absence of spasticity, the most constant and, next to atrophy, the most conspicuous feature of amyotrophic lateral sclerosis, permits us to say that this disease can be excluded.

Taking all these facts into consideration and giving due weight to the nystagmus, optic atrophy, absence of the abdominal and epigastric reflexes on the right side, and intention tremor, we are forced to the conclusion that the diagnosis of disseminated sclerosis is the only one that can be successfully maintained.

RECENT METHODS FOR RECOGNITION OF THE DIFFERENT TYPES OF HYDROCEPHALUS, WITH DEMONSTRATION OF A PATIENT WITH HYDROCEPHALUS DUE TO HYPERSECRETION

CHARLES A. ELSBERG, M.D.

A few years ago, we were satisfied with the diagnosis "internal hydrocephalus"; now we demand to know whether the case is one of obstruction in the aqueduct of Sylvius or in the foramina of Magendie and Luschka, causing an *obstructive hydrocephalus,* or whether there is no obstruction to the exit of cerebrospinal fluid from the ventricles into the subarachnoid space. In the latter class of cases, the distention of the ventricles by the fluid secreted by the choroid plexuses is due to diminished absorption from the subarachnoid space—*open hydrocephalus due to hypo-absorption*—or to increased formation of fluid—*open hydrocephalus due to hypersecretion* —or to a combination of hypersecretion and hypo-absorption.

Given a patient with hydrocephalus, we make the tests in the following manner: by lumbar puncture, 1 c.c. of cerebrospinal fluid is withdrawn, and 1 c.c. of sterile *neutral* phenolphthalein is injected. The sterile neutral phenolphthalein can be obtained in ampoules ready for use. If absorption from the subarachnoid space is normal, the first evidence of the dye should appear in the urine in ten to fifteen minutes, and 35 to 60 per cent should be

excreted in the urine in two hours (as tested by the colorimeter). If less than 30 per cent is excreted, the absorption from the subarachnoid space is diminished. In hypoabsorption by the arachnoid villi, the values most often obtained lie between 2 per cent and 14 per cent. In this way we learn whether absorption from the subarachnoid space is normal or diminished.

The next step is to determine whether or not there is free communication between the ventricles and the subarachnoid space. For this purpose, 1 c.c. of neutral phenolphthalein is injected into a lateral ventricle. In infants, this can be done through the lateral angle of the anterior fontanelle. In older children a small trephine opening must be made near the median line and just behind the coronal suture, under general anesthesia. After two minutes, a lumbar puncture is performed, and a few drops of cerebrospinal fluid are allowed to escape every minute and are tested. Normally, the phenolphthalein should appear in the fluid obtained by lumbar puncture within one to three minutes. If the dye fails to appear in the lumbar puncture fluid within ten minutes, the case is one of obstructive hydrocephalus, and if the little patient is under anesthesia, a puncture of the corpus callosum should be performed through the trephine opening already made.

If the examinations above described have shown that the normal communication between the ventricles and the subarachnoid space exists, and that there is either a normal or subnormal absorption of fluid from the arachnoid, the next procedure is to determine whether the cerebrospinal fluid secretion is greater than normal. For this purpose, a lumbar puncture is performed every day or two. When the secretion of fluid is normal, the quantity of fluid obtained at each puncture and the pressure of

the fluid become steadily less after the first two or three punctures, so that thereafter only small quantities, escaping drop by drop, can be obtained. In hypersecretion, however, the amount of fluid obtained and the pressure under which it escapes remain constant for long periods. Thus we have had under observation a boy of six years in whom 15 to 50 c.c. of cerebrospinal fluid were obtained every other day for several months.

Laboratory Findings.—In the little patient, herewith presented, in whom these tests were made, the following results were obtained: Forty per cent of the phenolphthalein injected by lumbar puncture appeared in the urine in two hours. After injection of the dye into one lateral ventricle through a small trephine opening, the dye was recovered from the fluid obtained by lumbar puncture within two minutes. By repeated lumbar punctures, large quantities of fluid (30 to 50 c.c.) under increased pressure were obtained every day or two. It was clear from these tests that the patient had a non-obstructive internal hydrocephalus, due to hypersecretion by the choroid plexuses. Of his treatment I shall speak presently.

In about 25 per cent of the patients with idiopathic hydrocephalus whom we have examined, the distention of the ventricles was due to obstruction, and a considerable number of these patients have been much improved or completely cured by puncture of the corpus callosum. In 15 per cent, the distention of the ventricles was due to hypersecretion, and these patients have had a lumbar puncture with removal of 20 to 50 c.c. of fluid every few days, combined with the internal administration of thyroid extract in increasing doses up to the physiological limit. From the experiments of Frazier and Peet we have learned that thyroid extract is the only known sub-

stance that decreases the amount of fluid secreted by the choroid plexuses.

Subsequent History.—The case which I have presented above has been treated by repeated lumbar punctures and the internal administration of thyroid extract. He has slowly improved and the improvement is continuing. Examination shows the boy no long ataxic; he is now free from headaches and vomiting, and the papilledema, which he had before the treatment was begun, has entirely subsided. During the past two years, we have seen some very satisfactory results from the combined lumbar puncture and thyroid treatment. These results, I feel sure, are much superior to those we had previously obtained by lumbar punctures alone.

In our experience, hydrocephalus due to hypo-absorption forms about 60 per cent of the cases. The treatment of this variety is very unsatisfactory and we have seen little if any improvement in our patients. We have not tried to drain the fluid into any cavities of the body, as I am not convinced that real success can be expected from any of the methods that have been devised. We must hope that the future will bring forth new and improved procedures. It is unfortunate that for this most common class of internal hydrocephalus, there is no good method of treatment. Thyroid extract and repeated lumbar punctures may be tried, but our results with this treatment have been poor.

In chronic hydrocephalus, the size of the head will always remain larger than normal, although its circumference may diminish considerably if the treatment is successful. I have seen the circumference of the head decrease from 4 to 8 cm. within two months after callosal puncture for obstructive hydrocephalus. If there is papilledema, this will rapidly subside under successful

treatment, and the improvement in the eye grounds and in vision will begin within a few days. If the ataxia has been of long standing, it may require years for its complete disappearance, although a marked improvement will occur within the first few weeks of the operation.

Many patients are brought to the surgeon with very advanced hydrocephalus. The blind spastic infants with enormous heads, in whom there is complete optic atrophy, and in whom there remains only a thin layer of brain cortex, are, of course, beyond all help. The earlier the patients are sent to the surgeon, the better the chance of relief by surgical procedures, after the type of hydrocephalus has been determined.

MYASTHENIA GRAVIS

JOSEPH COLLINS, M.D.

In the forty years that have elapsed since Samuel Wilks published, in *Guy's Hospital Reports,* the first case of the disease now generally known as myasthenia gravis, the disease has slowly yielded the secrets of its anatomical dependency, and to-day it may be said that it seems quite likely that it is in reality a disease of the striate muscular system, whose pathogenesis is immediately conditioned by disorder of one or more of the ductless glands. It is not without the realm of probability that the original development of the muscular system stands in relationship to certain internal secretions, and that disorder of this constitutes the anlage of the disease. There is such an amount of clinical and anatomical evidence pointing to serious implication of various ductless glands in myasthenia gravis that it can no longer be disregarded. When Erb, in 1879, called particular attention to the remarkable association of symptoms which constitute the disease, he conjectured that it was dependent upon a lesion in the upper portion of the oblongata; but the publication of Oppenheim's case in 1887 showed that the oblongata and, indeed, the central nervous system were quite intact. It was not until 1901, when Weigert published the report of a case in which a sarcoma of the thymus was found, and an infiltration of the diaphragm and deltoid muscles by small round cells not to be distinguished from those normally found in the thymus, that

the morbid anatomy of the disease began first to be built up. A few years before, namely, in 1898, Goldflam, who made one of the most important early contributions to the entire subject, had found in bits of the excised deltoid muscle cellular infiltrations similar to those described by Weigert. In 1904 Henry Hun published a case in which a careful examination of the nervous system revealed no lesion of it, but in which the muscles showed numerous foci of infiltration with lymphoid cells, and the thymus gland was the seat of a lymphosarcoma. Since that time many writers, and particularly Link, Hudlmosen, Burr, Buzzard, Mandelbaum and Celler, have published cases in which abnormality of the thymus has been demonstrated.

But there have been found in other instances definite lesions of the parathyroid (Chvostek), the hypophysis (Tilney) (Marinesco), and the thyroid (Boudon); and Landouzy and Sézary have published a case which they believed to be due to suprarenal insufficiency, in so much as the patient recovered while taking an extract of the suprarenal gland.

Of the cases that I present to-day, one of them shows profound disorder of the thyroid, and one has made and maintained remarkable improvement under the administration of thyroid and hypophyseal extract.

The diagnosis of myasthenia gravis is usually to be made with readiness and certainty, but one encounters in a large neurological clinic, cases that can by no means be diagnosticated either readily or satisfactorily. Myasthenia of arteriosclerosis, myasthenia as a manifestation of an occupation exhaustion neurosis, myasthenia of occult progressive muscular dystrophy, myasthenia accompanying hyperthyroidism without the distinctive features of Graves' disease, must all be differentiated from true

myasthenia gravis in certain instances. I have at the present time under observation a number of such instances.

The first patient is a girl who came to the Neurological Institute in April, 1916. Her complaint was a difficulty in walking. She walked very slowly because she had to drag her legs and they felt very weak. She experienced difficulty in carrying anything in her hands because of pain and a feeling of weakness which such effort caused in the upper extremities; difficulty in combing the hair, and in brief, in doing anything that required muscular effort. Her story was that she was well until the age of fifteen, when she first noticed difficulty in walking. She was going up the steps of the schoolhouse when she felt that she could not mount them all. She had been apprehensive that she was not going to pass the final school examinations which were then about to take place, and she attributed this weakness to the nervousness thus engendered. She passed her examinations, and during the summer of 1916 she said she felt quite well, but in October of the same year she began to complain of general lassitude. When she reached the school she could scarcely drag her feet up the steps, and when she persisted in doing so she experienced pain in the legs. About the same time she noticed that while eating, her jaws would get so tired that she would have to stop, and sometimes she would have to remove the bits of food from the mouth with the finger. Often during the night she would awaken and find that the tongue was between the teeth, always on the right side. About this time it was noticed that while playing the piano the ring finger of the right hand would fail to respond to her volition and gradually flexed on the palm, and as she persisted in the efforts of playing, the hand would become so heavy that it would not move. Gradually the strength of the

legs seemed to fail, and in November she noted that when she attempted to get on a car or step on a curb, her knees would oftentimes give way and she would fall. About the same time, in November, 1916, her family remarked that one of her eyes was so small that they thought she should go to a hospital for treatment. She relates that they examined her eyes and made an analysis of the urine at the hospital, and finding no abnormalities ordered her to take iodid of potassium up to forty grains a day. While she was taking this she grew rapidly worse and became so infirm that she could not walk, and her arms were so weak that she could not put a skirt on without assistance. This condition persisted for about six months, the symptoms not remaining of the same severity, but subject to variations from time to time. In March, 1917, she developed a new symptom, pain in the head, over the eyes. In May, 1917, she began to see double for the first time and to complain that the vision was blurred. She saw double on arising, but this would gradually disappear after she was up for a time. Off and on after the winter of 1916 she noticed at times that her heart would beat violently, particularly after excitement, but it was not until the spring of 1917 that she complained of an excessive sweating sensation, of warmth, and it was noted by many that her eyeballs were prominent. In July, 1917, it was remarked that her neck was swollen, and she complained that she often choked and had coughing spells on attempting to swallow liquids. There was no particular change in her symptoms otherwise, save that at times the myasthesia would become so profound that she could scarcely get about, but after resting for a few days she would feel about as well as usual. In September a new symptom was added, that of vomiting occasionally; and the manifestations of hyperthyroidism became more

pronounced. She came frequently to the Outpatient Department of the hospital until the latter part of September, 1917.

Physical Examination.—In April, 1916, when she was first examined, it was noted that she was a well-built, well-nourished girl, whose general appearance presented no indications of the disease. The facial expression was normal, as was the mobility of the eyes, face, lips, tongue and head. Speech was slightly nasal, but there was no difficulty in swallowing. Her station was secure, and her gait showed no distinct alteration. When she attempted to rise from a prone posture she got up in the way typical of a patient with muscular dystrophy. The grip in both hands was good, but there was a pronounced weakness of the muscles of both shoulder girdles, although she could put her arms in any position. Direct elevation of the upper extremities was accomplished with much effort, and to maintain them there for longer than a few seconds was difficult. There was marked weakness of the lumbar and iliopsoas muscles. She was unable to raise either leg at a right angle to her trunk, or to rise from a prone posture without rolling over on the side or pushing herself up with the arm. Adduction of the thigh was feeble.

The superficial and deep reflexes were normal. There were no sensory disturbances and no indications of muscular atrophy or hypertrophy. She was taken into the hospital at this time and kept under observation for a week, during which period examinations were made of blood, gastro-intestinal contents, urine, spinal fluid and so forth, but nothing abnormal was found. Her pulse rate was not accelerated, save on physical effort, but her blood pressure was constantly low (systolic 110, diastolic 80).

Diagnosis.—At that time it was thought that she was a typical case of progressive muscular dystrophy, although she complained of symptoms referable to the muscles supplied by the pontobulbar nerves. There was no evidence, while in the hospital, that they were disordered. In other words, she had no objective findings referable to the third, fifth, sixth, seventh, ninth, tenth, or other cranial nerves that are habitually disordered in myasthenia gravis. In the early winter of 1916, however, when she was next seen, the manifestations of myasthenia gravis had become so distinct that there was no hesitancy in considering it an example of that disease, although even then the objective cranial nerve symptoms which we habitually see in such cases were trifling. The myasthenia was displayed most profoundly in the extremities and in the trunk. She could not do her hair, she had difficulty in putting a vest over her head, she frequently would drop her knife and fork, she began to waddle when she walked a short distance, and she frequently fell when she attempted to step up. It was at this time that we were able to satisfy ourselves for the first time of the typical myasthenic electrical reaction which was first described by Jolly, an exhaustibility of the tetanizing faradic of the nerve or muscle, the muscle contraction growing weaker with each stimulation, until it is finally lost, but is regained after the muscle has had a rest—usually of about a minute.

In the spring of 1917, when she again returned to the clinic (she had meanwhile been under treatment at one of the eye hospitals), the clinical picture was typical of myasthenia gravis, and it continued to be so until early in July, 1917, when the customary symptoms of hyperthyroidism, namely, enlargement of the thyroid gland, a moderate degree of exophthalmos, hyperhidrosis, and

caumesthesia were added. The occurrence of these objective symptoms did not seem to coincide with any increase of the myasthenia symptoms; in fact, when she was last seen on September 14th, 1917, it was noted that she thought her hands and arms were improved. She was able to fix her hair and to do many things with her upper extremities which she had not been able to do. The strength of her lower extremities was no better. She did not complain of diplopia save occasionally on arising, and she had not latterly remarked ocular ptosis. She was demonstrated with three other patients with myasthenia gravis to the military medical officers on duty at the hospital on September 26th.

Within a few days after this time she went of her own initiative to one of the ophthalmic hospitals, where she was counseled to have an operation on her thyroid gland for the relief of the disease, which was considered Graves' disease. Her superior thyroid of the right side was ligated and she died within a few hours.

The Clinical Picture.—When Hoppe of Cincinnati in 1892 fixed the clinical picture of myasthenia gravis, or as it was then called, "bulbar paralysis without anatomical foundation," he pointed out that in addition to the fact that the disease reminded one forcibly of true bulbar paralysis, its most striking characteristics were the absence of muscular atrophy, the participation of the upper part of the face and the motor oculi nerve in the display of the paralysis, the absence of disorder of the hypoglossal and the tendency of the disease to display itself in exacerbations and remissions which later might be so considerable as to constitute an approach to recovery. It was Goldflam, however, who emphasized the fact that asthenia is the dominant note of the clinical picture, asthenia and a profoundly increased fatigability of the

muscles to any form of excitation. This myasthenia may display itself in any part of the body, but it is much more likely to show itself first and most conspicuously in the domain of the cranial nerves, and particularly in that of the third, seventh and ninth. However, in one of the cases that I publish herewith, the myasthenia exhibited itself in the extremities for more than a year before there were any indications of its occurrence in the muscles supplied by the cranial nerves. Usually the first symptom is some disorder of the motor oculi. The patient complains of seeing double, and that the lids are weary and droop. The symptoms may confine themselves to such ocular display for a long time, months, even years, but as a rule they but briefly precede the occurrence of difficulty in chewing, of performing movement requiring dexterity and celerity of the tongue and lips, and finally of all the subjective symptoms of bulbar paralysis. When the disease is in full display we have a clinical picture made up of partial or complete ptosis, of an intensity which often varies within the day, and sometimes within the hour, of some form of external ophthalmoplegia; of a variable degree of bilateral facial paralysis; of implication of muscles that subserve articulation, mastication and deglutition; weakness of the muscles of the throat and neck, which make it difficult for the individual to maintain the head in its ordinary position without effort; of the muscles of the extremities, which interfere with the execution, sometimes, of even the simplest acts; of the muscles of respiration and of the abdomen, which reveal their participation in the asthenia by dyspnea, ballooning of the abdomen, and by collapse phenomena.

The asthenia varies in intensity at different times up to the most complete paralysis. One may observe its display and the increasing impotency of the muscle to re-

spond to stimulus, by observing the muscle react to volitional or to electrical stimulus.

The remarkable feature of the disease is that despite the well defined disease of the muscles often found after death, true muscular atrophy has been rarely observed. It does result, however, and I am of the opinion that it will be noted more frequently in the future, especially in cases that have been under observation for a long time. In the first case in American literature reported by me, 1896 (*International Medical Magazine*), the patient developed, after she had been under my observation for 10 years, a decided atrophy of the pelvic girdle and anterior tibial muscles, so that her gait, posture, and attempts at rising at all times were like those of an individual ill of muscular atrophy. There are a number of these cases in the literature, a typical one being that reported by Fabris (*Riforma Medica,* 1907, page 988), in which there was atrophy of the muscles of the neck, of the shoulders and the arms.

It is frequently written that the sensory sphere is spared, and so it is in the majority of instances, but many of the patients complain of headache, some of them of neuralgic pains in different parts of the body, others of paresthesiæ, and many of unutterable malaise. The pains are sometimes postural pains. For instance, in Goldflam's patient who had severe pain in the neck, it might well be conceived that this pain was the result of strain to keep the head erect. In some cases, such as those of Hun, the pains of which the patients complained might similarly be interpreted. In the case reported by Ballet, in which there were trigeminal neuralgia and a complete asthesia to touch, temperature and pain, it is legitimate to assume that there must have been coëxisting disease of the Gasserian ganglion. In one of Buzzard's cases

there was an analgesia and anesthesia in the ulnar half of each arm. On the trunk and legs there were æsthesia and analgesia, resembling that of a well marked case of tabes, and the patient had complained of "lightning" pains. It has been contended that these sensory phenomena militate most seriously against the acceptance of the theory that the disease is of the muscular rather than of the nervous system; but pain, paresthesia and objective sensory disturbance often accompany myositis.

Disorder of the senses of taste, smell and sight have all been noted occasionally. In a disease so overwhelming and prostrating, it is understandable that exhaustion manifestations may be revealed in the tensor tympani and muscles of accommodation.

Disorder of the Emotional and Mental Spheres.—Many cases of myasthenia gravis are diagnosticated "hysteria" or "nervous prostration" by the general practitioner, particularly in the early display of the disease. This is largely due to the fact that the patient makes complaint of serious symptoms referable to organs that seem to be normal on examination, and because they profess a prostration not in keeping with the findings of physical examination. It is a mistake to assume that mental and emotional disorders do not occur as an integral part of the disease. In the case that I published in 1896 the patient was subject to attacks of mental confusion. One of Buzzard's patients had ideas of persecution, depression and homicidal thoughts. Mohr published a case subject to excitement; Raymond and Alquier a case in which there were depression of spirits and loss of memory; Goldflam a case in which there were epileptic attacks; Gowers a similar case.

The disease in full display constitutes a clinical picture as characteristic and readily recognizable as paralysis

agitans. The facial expression of the disease is adequately depicted in Fig. 3 (page 263). The bodily manifestations naturally depend upon the intensity and extent of the paralytic phenomena. The physical accompaniments of the disease, aside from the obvious paralysis, are practically nil, save a reaction of the nerves and muscles to the faradic current, which is known as the myasthenic reaction. The muscles and nerves rapidly lose their faradic excitability after a number of contractions, sometimes 50, sometimes 150, and then after a rest they regain it.

In the morning, on awakening, the muscles usually functionate fairly well, then little by little ptosis, strabismus, facial diplegia appear, followed by difficulty in the performance of any or all voluntary movements. The asthenia of the respiratory muscles, which may be delayed for a long time, shows itself in dyspnea, which often comes in crises of great severity. That of the heart muscles shows itself in tachycardia, sometimes in asystole and intermittency of the pulse, and lowered blood pressure. The fatigability of the muscles of the iris was pointed out by Grocco, by Link, and by others.

In some cases, however, the symptoms are continuous. In the case that I reported in 1896, the patient did her work for 10 years. Though the myasthenia existed constantly, it overwhelmed her only at the menstrual periods. After it had been in existence for about 12 years she became so weak and infirm that she could scarcely move, and when she did, attacks of dyspnea and palpitation occurred. There is a type of myasthenia gravis that progresses uniformly, month by month, and leads gradually to dissolution, usually within three or four years from the time of its onset. Whether this variety of myasthenia is different from the ordinary myasthenia gravis remains to be seen.

The symptoms of myasthenia gravis sometimes apparently display themselves abruptly, and reach a full degree of development, as it were, within a very short time. This was so in the case of a priest, 37 years old, in whom the symptoms had been in existence for eight months when he came first under observation. He considered himself in most perfect health, when he remarked one evening while playing the piano that the ring finger of the right hand became stiff and unwieldy. After he stopped playing it seemed to be all right. A few weeks later he remarked that the entire right forearm would display this same feeling of stiffness and weakness. He did not give particular heed to these occurrences until a month or so later, when he remarked that his face would become very tired after chewing a little while, and that he often had to hold the food in the mouth for a short time after chewing it, before he could swallow it. For some time he had great difficulty in swallowing, even when he swallowed very slowly, and occasionally the food would go down the wrong way and provoke the most distressing coughing spells. These symptoms existed off and on for two months before he first saw double, and before members of his household noticed that his eyelids would tend to close at times. Now and then the myasthenia would come upon him in an overwhelming way. It would produce at times a very dramatic effect, such as at the moment when he was elevating the Host in the celebration of the Mass, or when he was ascending the steps of the altar. It was likely to come at any time when he hurried, for instance, to catch a car; and oftentimes his knees gave way, and he fell. In its full display the myasthenia was quite classical. There were no ancillary phenomena which many of our patients have had, such as headache, vertigo, paresthesia, ballooning

of the abdomen and vasomotor manifestations. When the myasthenia was most pronounced, he felt a decided sense of prostration, and the extremities were cold and sometimes clammy. There were no mental or emotional manifestations or symptoms. He treated what seemed to be a situation of great gravity, with much equanimity.

Physical Examination.—The physical examination revealed nothing in any way out of the ordinary, save that the shape of the pupils was slightly irregular, and that the light response was not very prompt. This was corroborated several times by Dr. W. A. Holden. He likewise had inconstant, sometimes well marked, nystagmus of both eyes. He complained of diplopia in extreme directions of the gaze, and the left ptosis which he displayed was variable. The myasthenic electrical reactions were by no means well defined. The facial muscles and the muscles of the extremities responded well and quickly up to two hundred sequential contractions, without revealing the so-called myasthenic reaction. It has been my experience that the myasthenic reaction is neither so constant nor so diagnostic as writers on the subject would have us believe. I am convinced that it is by no means a necessary accompaniment of the disease. This patient has been under observation for upward of a year, and at no time has the myasthenic reaction been clearly demonstrable. The laboratory examinations were negative throughout.

An interesting feature of this case is that the patient had always been a big eater, and that he was consequently fat and gross. Although only five feet six inches in height before the illness developed, he weighed nearly 200 pounds. He improved very materially while taking a combination of extract of thyroid and pituitary, and

Case of Myasthenia Gravis, Showing Affection of Eyes.
Patient—A priest thirty-seven years of age.

after the use of the *x*-ray over the upper part of the sternum.

Perhaps the most typical case that we have recently had under observation is that of a young married man, 24 years old, who relates that when he was about twenty he noticed that his fingers got weak in turning the key in the door. Soon after that, diplopia developed, and the first attack lasted two months. He went to an ophthalmologist, who prescribed glasses, but the wearing of them seemed to make the diplopia worse. The next symptom to develop was a weakness of the hands and the upper extremities, especially manifest when he attempted to carry anything. It did not seem to matter how light the object was, his hands would get so tired that the object, for instance, a light cane, would drop out of them. One of these incidents which he recalls, occurred about nine months after the onset of his trouble, and when he consulted a physician, he was told that he had a general nervous breakdown. About eighteen months after the symptoms first showed themselves, he had an attack of bronchial pneumonia, and afterwards went to a convalescent home for a month. When he left there he got a job as a conductor on a Fifth Avenue bus, but he had to give it up because he found that he could not push the bell and that he could not register the fares. After this the myasthenia involved all the extremities and the face and head. He had difficulty in standing and walking, difficulty in using the hands, putting on his clothes, feeding himself; difficulty in chewing, swallowing, speaking; and in brief, in all purposeful movements. The myasthenia is subject to great variation of intensity, and it can always be induced by making effort.

Physical Examination.—The physical examination revealed nothing particularly noteworthy, aside from the

general facial features of the disease, which are well illustrated by the accompanying photograph. There were many somatic features of what are commonly believed to be disorder of the internal secretions. The hair on the body was of a typical feminine distribution, the hands were big, the fingers long and tapering, the jaw was prognathous, the lips thick, and the general cast of the features leonine. The tendon-jerks were lively and did not display any tendency to exhaust on fifty sequential repetitions. There was a definite myasthenic reaction in the facial muscles to the electrical current.

The course of the disease was subject to great variation. On some days he felt too weak and ill to move, on other days he felt fairly well. He himself attributed some relation of the disease to a fall from a truck, which he had had about six months before the first symptom displayed itself. He landed on the end of his spine and was unable to straighten up for about a quarter of an hour. The next day he did not have any particular symptoms that he remembers. His symptoms became very much worse two years ago, immediately after he received a shock from the accidental death of a brother. This patient was under treatment for several months by the general physical treatment of administration of extracts of the ductless glands and by *x*-ray applications over the upper sternum region, but no improvement was noted.

Now and then we encounter an incidence in which the diagnosis of myasthenia gravis cannot be definitely established, even though that seems to be the most probable diagnosis. Such a case I had under observation for a considerable period in December, 1915.

A married Jewess, thirty-eight years old, complained of inability to raise the eyelids, the left being almost immobile; of weakness and an agitated feeling in the lower

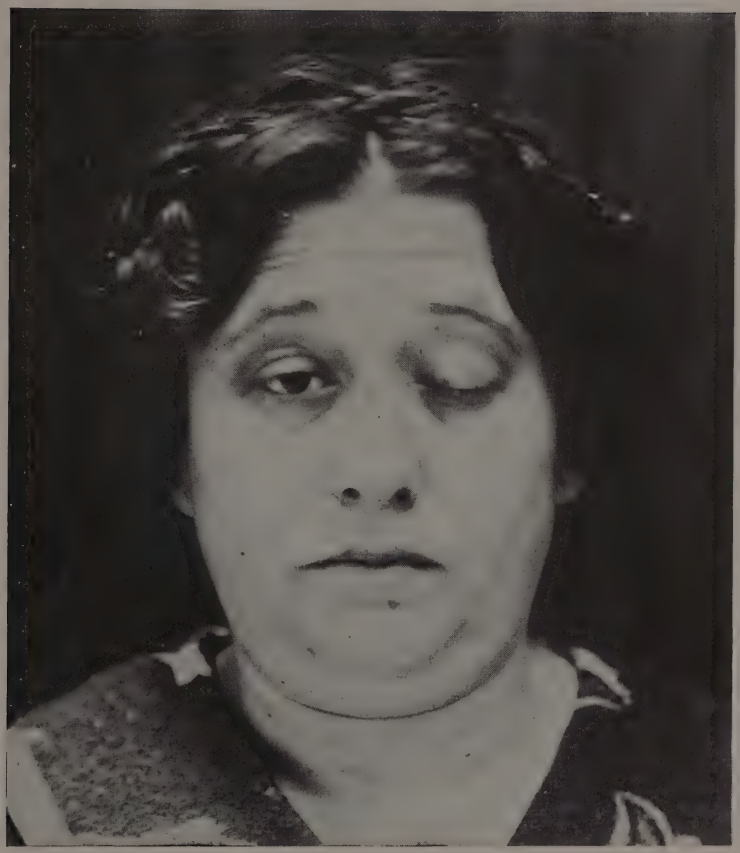
Typical Case of Myasthenia Gravis.
Patient is twenty-four years of age.

extremities; of a sensation of coldness through the lower part of the body and legs; a sensation as if she were going to fall when she stood or walked; a sensation of loss of strength in the upper extremities; of difficulty in chewing at times, and of enfeeblement of the jaws. These symptoms had come on abruptly two months before she came to the clinic, and those referable to the eyelids had existed a month before the weakness of the extremities showed itself.

Her history in detail was that in the winter of 1914 she had had what was called an attack of grippe. The symptoms were headache, chilliness, fever, stiffness of the neck muscles, sticking pain all over the body, and what might be called a generalized tenderness and immobility such that it hurt her to have her skin touched anywhere, and she was unable to close her hands. She had to be turned over in bed and could not stand on her feet on account of weakness; when she attempted to do so, pin and needle sensation developed to a most distressing degree. She spent the entire winter in bed, and the following June went to Mount Clemens, where she remained for a month and was very much improved by the treatment. The following autumn, September, 1915, she developed severe headaches, particularly in the forehead and temples, and was often drowsy and would fall asleep at any time of the day. At night she was sleepless because the headache was so severe, and these headaches continued until November, when the left eyelid began to close, and shortly after this, the right eyelid became weak. At this time the Wassermann reactions of the blood serum and cerebrospinal fluid were quite negative, as was also the *x*-ray of the skull. She was treated by the administration of iodid of potassium, but she seemed to get worse while taking it; particularly the strength in the

upper extremities became impaired, her hands and arms becoming so weak that she could not hold things in the hand—for instance, a knife and fork. Eight weeks previous to her coming to the Neurological Institute, she had noticed that the jaw muscles grew tired while chewing, she had not remarked difficulty in swallowing, but her voice had become weak and often hoarse. It was during these two months that she had noticed increasing unsteadiness of station and gait, and had fallen several times on attempting to walk. In addition to this she had become very emotional, and in particular had crying spells and periods of agitation.

Physical Examination.—The physical examination at this time showed a large obese woman whose facial expression is illustrated by the accompanying photograph. When it was taken she was forcibly endeavoring to open the eye. Her station and gait were unsteady, and there was distinct weakness of all the muscles of the upper extremities, most marked in the extensors of the arms. She was unable to keep her arms extended when the slightest force to flex them was applied by the examiner. There was no muscular atrophy. The tendon-jerks were all present; those of the lower extremities were sluggish. There was no Babinski phenomena; the abdominals and epigastrics were elicited only with difficulty, as is often the case in individuals who have large and pendulous abdomens. There was no distinct sensory disturbance, although the patient seemed more sensitive to contact and to pin prick on the outer surfaces of the arms than on the inner, and more sensitive between the fourth and eighth thoracic spines than above and below them. There was no tenderness of the muscles on deep pressure. All the muscles of the upper extremities responded promptly and actively to both faradism and galvanism,



Case Diagnosticated as Myasthenia Gravis, Illustrating Facial Expression.

Patient is a woman, thirty-eight years of age.

and there was no appreciable difference in the response of the two sides. There is no suggestion in the reaction of the so-called myasthenic reaction. For instance, both tibial and both triceps contract 120 times without appreciable sign of fatigue. Examination of the eyes showed that the right pupil was 3 mm. in diameter, the left 2½. The right was slightly irregular and the left oval. Both responded to light and convergence. The fundi were normal; both corneals showed many opacities. There was a bilateral ptosis, and the mobility of both eyes was very limited. There was much restriction in the movements of the left, outward, upward and downward. In fact there was very little extra-ocular movement remaining. In the right eye the movements were limited outward, upward and downward. All the laboratory examinations were negative. Two months after the notes of this examination were made, it was recorded that there had been a decided change in her emotionally, but very little physically. She was inclined to be obstreperous, combative, notional—in brief, what is commonly called hysterical; and these moods affected her physical condition. An effort was made to use general therapeutic calisthenics-massage, Zander exercises, tonic baths, and so forth. It was necessary to discontinue them because she would fall abruptly, wherever she happened to be. At times it would seem as though this was an affectation, because she would apparently pick out the most conspicuous positions and dramatic situations, such as in front of the ward full of people, or in a corridor where there were strangers, or before the attending physician. Later these falls became so frequent and her weakness so severe, that she had to be allowed to remain in bed. The most striking symptom was the celerity with which muscular fatigue occurred. The strength of the extremities seemed to vary from day

to day. One day she appeared to have considerable power in the hands and arms; the following day she would be emotional and "hysterical" and persist that she was getting weaker every day. This would appear to be the case; then the next day, or a few days later, she would have an adequate amount of strength to go about in a halting, rather infirm way. The ptosis of the left eye, indeed of both eyes, gradually lessened in intensity, but never disappeared. She regained considerable power in the movement of the left eye, especially upward, downward and inward movements. A few days later it was noted that she was profoundly depressed, crying and insisting that she was not getting well and wanted to go home. The ptosis in the left eye was again complete, that of the right eye being more marked than it was before. The range of the ocular movements was much more limited in all directions than it had been a week before. The general physical strength was about the same as on previous examinations. At this time it was noted that the tendon-jerks of the lower extremities, and of the upper as well, tended to become exhausted on repeated sequential elicitation. For the first time it was noted that the facial muscles seemed to present the beginning of a myasthenic reaction.

Diagnosis.—In view of the peculiar manifestations and distribution of the muscular display, the onset with headache, and particularly in view of the variance of her symptoms from week to week, the diagnosis of myasthenia gravis seemed the most likely one. It was realized, however, that it was open to criticism, because of the absence of the myasthenic reaction and the difficulty in establishing the genuine muscular fatigability. To be sure, there was readily demonstrated fatigability of the muscles, but it impressed one as the fatigue that would

accompany a very mild degree of neuritis. It was impossible to assume that we were dealing with a pituitary or pineal neoplasm, in view of the absence of any distinctive symptoms of these disorders and the negative *x*-ray findings. Then the emotional symptoms that she displayed were not readily interpreted. She was unamenable to suggestion or discipline, she was incorrigible, she was a menace to the comfort and welfare of the other patients in the ward, because of her constant complaint and agitation; and still her behavior was in no way characteristic of what is properly termed mental disorder. For instance, as an illustration, it was impossible to test her sugar tolerance, because of her refusal to coöperate and because of her contempt for the belief that it might throw some light on the nature of her disorder.

Causation and Pathogenesis.—Upwards of 50 per cent of all the cases reported have been between 20 and 35 years old. It has been said that women are more liable to it than men, but there is very little sex influence. A very few cases have been reported in early childhood and a few after sixty. In reality it is a disease of early adult life. In view of Harlow Brooke's convincing contribution to the subject of myasthenia (*The Journal of Medical Research,* XXVII, January, 1913), in which it is shown that the symptoms of myasthenia gravis may be dependent upon a visceral arteriocapillary fibrosis, chiefly involving the medulla oblongata, I hesitate to accept as genuine instances of myasthenia gravis reported as occurring in individuals above fifty. The case reported by Dr. Brooke was a fairly typical one of myasthenia gravis, as I had the patient under observation for a considerable time.

The most interesting thing to me in the etiology of the disease, so far as I have observed it, is its relation to

fright, worry, trauma, and so forth. The ease and readiness with which these factors upset the internal secretions, especially the suprarenal and the thyroid, and possibly all of them, immediately come to mind.

The theories of the pathogenesis of myasthenia gravis are, first, that it is a disease of the nervous system, the chief symptoms being displayed in the realm of the pontobulbocervical nerves. As these nerves have been found intact in the cases that have come to autopsy, it has been assumed that the disease of them is one which inhibits their function without causing disintegration, and that one colossal inhibition of function has spelled death for the individual. The hypothesis has been advanced that this perversion of certain segments of the cerebral nervous system is caused by some poison circulating in the blood, and there have been a number of investigations undertaken to discover what this poison might be. So far, very little success has attended these efforts.

The second theory is that myasthenia gravis is in reality a disease of the muscles and not a nervous disease at all. There is much more to be said in favor of this view than of the former. The muscles of myasthenia gravis obtained from biopsy or necropsy have often been found decidedly diseased, and the pathological changes are of two kinds: (1) foci of infiltration around the blood vessels and in the interstices of the muscle fibers, constituted of lymphocytes and plasma cells, and (2) changes in the muscle fibers themselves, of the nature of simple atrophy, degenerative atrophy, fatty degeneration, plasmoidal retrogression, proliferation of the nuclei, of the sarcolemma, and the formation of pale fibers. Frugoni, the most recent and warmest advocate of the myogenic theory, claims that these alterations are specific of the disease. The serious objection to this theory

is that the characteristic exhaustibility of the muscles and their tonetic restitution does not fit in well with it. The "bulbar" manifestations may be explained by anatomical changes found in the diaphragm and other thoracic muscles, but the mechanism of the characteristic exhaustion does not fit in very well with the conception. However, it is a far more plausible theory than the neurogenic theory. It explains the ancillary but not essential symptoms of the disease, such as pains in the muscles, sensory disturbances, postural disorder and shrinkage of the muscles, better than does the other theory.

The third theory is that myasthenia gravis is a disorder of the neuromuscular mechanism constituted by functional perversion of the endocrine system, particularly of the thyroid and suprarenal, and that this functional perversion may eventuate in structural disorder, either of the nerves or of the muscles. In the first place, the frequency with which a lesion of the thymus has been found (20 per cent), the association of the syndrome with obvious thyroidal disease as in the third case of this series, and, what is by no means uncommon, the instances in which definite symptoms such as bronzing and pigmentation of the skin, pointing to disorder of the adrenals have been observed, and the other instances in which the ductless glands have been found diseased, are very suggestive.

It does not at all follow that the suprarenal gland of an individual who died of myasthenia gravis, functionated normally because it was found structurally normal at autopsy. The autopsy table eventually suggests the pathogenesis of myasthenia gravis, but the physiological laboratory is where the riddle is likely to be solved. In view of the highly suggestive work that has already been done by Cannon on the relation between depleted psychic

states, between depressed emotional states and the endocrine system, we may confidently look forward to an explanation of the disease from the experimental physiologist. The myogenic theory of the disease and the neuromyogenic become quite reconciled with this conception.

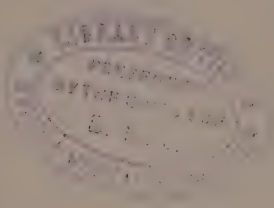
It need scarcely be said that no one subscribes to the view promulgated by Brissaud and Lentzenberg, that myasthenia gravis is a form of polioencephalomyelitis, or by Charcot and Marinesco, that it was due to the abolition of the motor functions of cells that still preserved their trophic function.

The diagnosis of the disease, when it displays itself in one of the more uncommon ways, is by no means easy. The young girl of the series herewith described was diagnosticated muscular dystrophy by a competent and experienced neurologist. It is to be remembered that in about 20 per cent of the cases the early symptoms display themselves in the extremities. In these cases the symptoms may be closely paralleled by occupation neurosis. A cigar maker under observation here in 1910 had a frequent, rapidly developing exhaustion of the upper extremities, amounting nearly to a paralysis, which was very suggestive of myasthenia gravis. Had the face been implicated, that diagnosis would surely have been made. I have likewise seen cases of myasthenia, unaccompanied by myokymia of a grave quality, develop after intensive physical training, and terminate in complete recovery.

I have had no experience with the clinical variety of the disease that is alleged to be of very acute onset. In all the cases of apparently sudden onset that I have seen, careful inquiry always has revealed the insidious development of the disease.

The treatment of myasthenia gravis is a grim subject to discuss. Some patients improve while extracts of vari-

ous ductless glands are being administered. Others improve without therapy, but most of them do not improve under any therapy. Several apparent cures have followed *x*-ray application to the region of the thymus. The justifiable therapy is the administration of the ductless glands and exposure of the ductless glands, that are thought to be the seat of disease, to the *x*-ray.



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